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THE ROENTGENOLOGIC DIFFERENTIAL DIAGNOSIS BETWEEN CANCER AND DIVERTICULITIS OF THE COLON[1]

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THE difficulty in differentiating diverticulitis of the colon, particularly of the sigmoid colon, from a carcinoma of the bowel is as old as the knowledge of diverticulitis as a disease. Though diverticula of the colon were known to anatomists and surgeons as accidental and rare findings, their clinical importance was not recognized before Graser's demonstration in 1898. He showed the comparative frequency of diverticula, the possibility of inflammatory processes being caused by them, and the difficulty of differentiating the inflammatory mass from a malignant growth of the colon.

In the following decades, a number of excellent clinical (Beer, Moynihan, W. J. Mayo, Telling and Gruner, Judd and Pollock, Spriggs and Marxer, Rankin and Brown, and others) and roentgenologic papers (Abbe, Case, Carman, George and Leonard, Spriggs and Marxer, Berg, Stewart and Illick, Golden, Singleton and Hall, and others) have decreased the differential diagnostic difficulties. Occasionally, however, there are still cases in which diverticulitis leads to such deformities as to make its differentiation from neoplastic disease extremely hard. In such a case one often asks oneself whether it is necessary to enter into an extensive differential diagnostic discussion or whether it would not be much better to transfer the responsibility to the

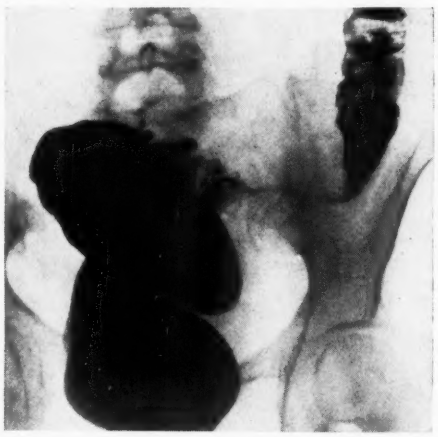


Fig. 1. Long area of narrowing in the sigmoid due to diverticulitis in a 62-year-old woman who had had attacks of low abdominal pain since the age of 17. Present attacks started six weeks previously with fever and pain. X-ray examination showed multiple diverticula proximal to the narrowing (only one diverticulum visible in this view). The narrowing had not changed essentially three weeks later. At some time during the x-ray examination diverticula were demonstrable within the narrowed area. One year later a perforated diverticulum was drained.

[1] Read before the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Nov. 28–Dec. 2, 1938.

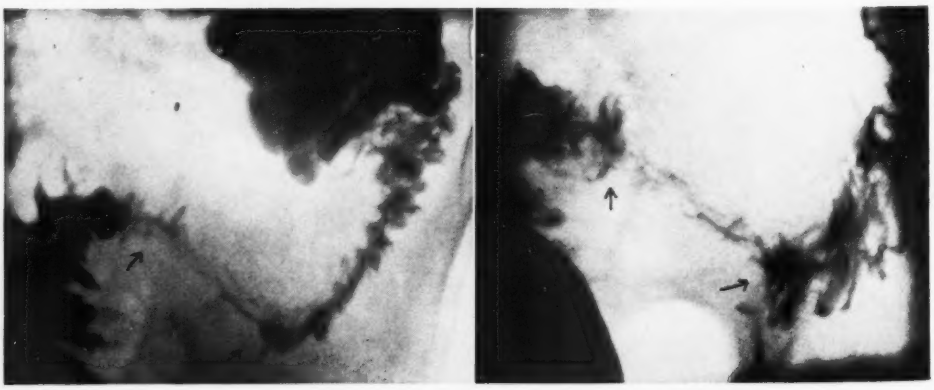
surgeon and have him solve the problem by exploratory laparotomy. Unfortunately, the diagnosis is by no means easy or even possible at operation in a fair percentage of cases.

Fig. 2-*A*. Small localized area of constant narrowing due to diverticulitis (*see arrow*). Shelf-like beginning and ending of this area. Preserved mucosal folds. Changing spastic areas proximal to the narrowing. For clinical data, see text.

Moynihan, in 1907, while discussing the mimicry of malignant disease in the large intestine, reported the story of a man with a long history of stomach trouble who had had, on two recent occasions, signs of intestinal obstruction which included fecal vomiting. At operation, a hard inflammatory mass was felt in the duodenum with evident duodenal obstruction. In the ileopelvic colon another hard mass was found which was thought to be a cancer. Since this was deemed to be the more important condition, the duodenal ulcer was left untreated, and the tumor was excised. The opened specimen showed no evidence of cancerous growth, but diverticulitis, the wall of the gut being in some parts one and one-half inches thick.

Mayo, Wilson, and Griffin, in the same year, reported five similar cases and stated: "It is impossible to tell, even by the gross appearance of such a tumor, whether it is inflammatory or malignant."

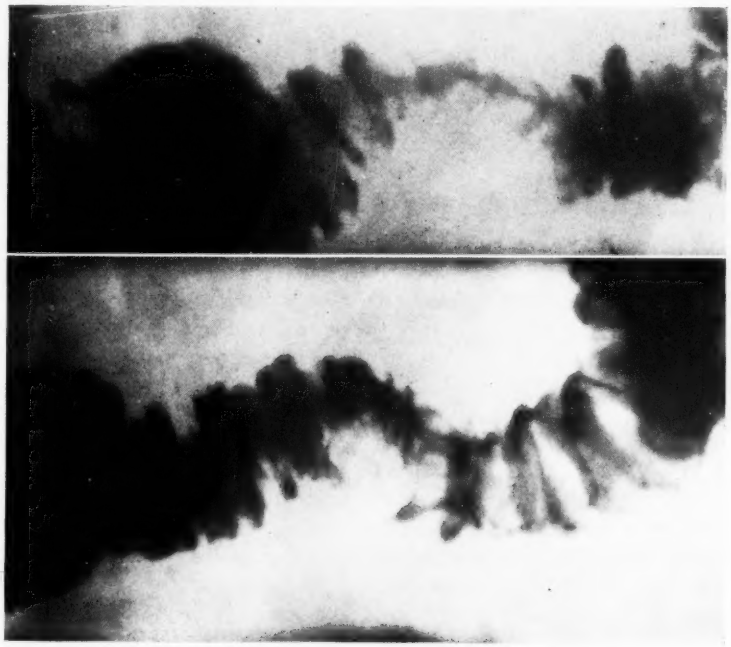
Florian described a patient having intermittent colic, attacks of fever, and intestinal hemorrhages, who had a defect of the sigmoid, shown by x-ray examination, which was called carcinoma. The surgeon had the impression that he had resected a carcinoma, even after he had the specimen in his hand. Microscopic examination alone showed that an area of chronic diverticulitis and not a cancer had been removed.



B *C*

Figs. 2-*B* and 2-*C*. Spot films of the narrowed areas shown in Figure 2-*A*.

Golden reported the history of a patient in whom the family physician had made the diagnosis of cancer of the descending colon based on the findings during a barium enema examination. A hard, non-tender mass was felt in the left lower quadrant. The patient was operated on immediately after his admission to the hospital without any further x-ray studies. At operation the mass was thought to be carcinoma, even though numerous diverticula of the descending colon and sigmoid were noted. The dissection of the specimen proved the mass to be due to diverticulitis and not to cancer.



Figs. 3-*A* and 3-*B*. *A*, Small fibrostenotic area in the sigmoid due to diverticulitis. Note preserved mucosal folds in the area of narrowing. *B*, Three months later the spasticity around the narrowing has improved; the narrowing itself is still present. Clinical data: 13 days previous to the first examination patient was operated on under the diagnosis of acute appendicitis. The operation showed diverticulitis of the sigmoid with perforation.

Among 18 cases of diverticulitis recently reported by Stetton and Abeloff, three had been called cancer at the time of operation. Two of these patients died following the operation. Similar cases have been reported by Telling, Judd, Coombs, and others. In fact, almost any abdominal surgeon can recall cases from his own experience.

A few cases taken from the records of our own hospital may further illustrate this problem.

A man, 54 years old, had been examined outside the hospital and the clinical diagnosis of cancer had been made. The x-ray examination, done on the outside, showed diverticula. At operation a mass was found in the region of the sigmoid. The surgeon was absolutely positive that it was carcinoma and thought it to be inoperable. No x-ray examination was done in our hospital when the patient entered it for a second operation. The mass in the sigmoid was confirmed and, at the time of the operation the differential diagnosis between cancer and diverticulitis was considered. The sigmoid was resected, the

specimen showed diverticulitis, with no evidence of cancer. The patient died post-operatively.

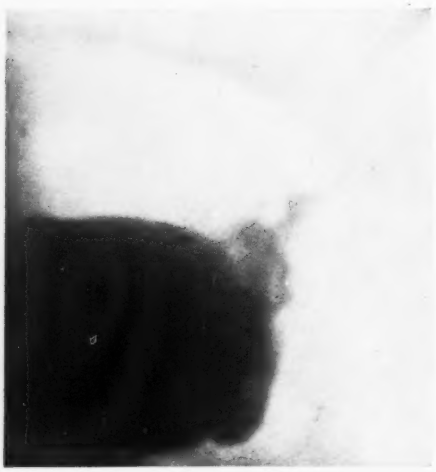


Fig. 4-*A*. Obstruction due to diverticulitis of the sigmoid. The film shows the tip of the barium enema where there is no tumor visible.

Examination of a woman, 36 years of age, with a nine-day history of a "bloated feeling" in the abdomen showed a large, slightly tender mass arising from the pelvis. At laparotomy a large fibroid of the uterus was removed. As the operation was being completed, a hard nodule was felt in the sigmoid. This seemed to be producing some constriction, and, as there was an implant on the peritoneal surface which looked malignant, a Mikulicz resection of that loop was done. The resected specimen showed a single diverticulum extending into the mesenteric attachment, surrounded by a fibrous mass, 3 cm. in diameter, and two firm glands close to the peritoneal surface of the mesentery. Microscopic examination proved the presence of chronic diverticulitis. There was no evidence of malignancy.

All these reports demonstrate dramatically how difficult—or even impossible—it may be to differentiate between diverticulitis and cancer during the operation.

Inasmuch as resection of diverticulitic masses is connected with a high operative mortality and is indicated in comparatively rare instances, the necessity of making the correct diagnosis before opening the abdomen is obvious. Clinical data in regard to age, weight loss, pain, stool habits, temperature, and blood picture are of some, though usually not decisive, importance. They will not be discussed in this paper (see Telling and Gruner, Judd and Pollock, Spriggs and Marxer, Case, Rankin and Brown). The presence of blood in the stools is strongly in favor of cancer in a questionable case, and I agree with those authors who advise looking for an additional source of hemorrhage if, in a case of diverticulitis, blood is found.

Unless proctoscopy demonstrates a cancer the responsibility of differentiating new-growth from diverticulitis rests heavily and almost exclusively on the roentgenologist. It is the main purpose of the present paper to stress this sometimes uncomfortable but unescapable responsibility which is recognized by competent clinicians, though the necessary consequences inherent to such a responsibility are not always followed by either roentgenologist or clinician in the handling of such a case.

The pathology of diverticulitis has been described in excellent reports (see Telling and Gruner, Spriggs and Marxer, Case). It will be mentioned here only inasmuch as it seems necessary for the differential diagnosis.

The inflammation within and around diverticula, probably due to stagnation of fecal material within them, leads to swelling of the mucosa. It is possible that some of the swelling is due to lymphostasis produced by sclerosing inflammatory processes of the mesenteric glands, as suggested by Berg, Pohl, and Fleischner. The course of the disease may be acute, like appendicitis, and may lead to perforation of a thin-walled diverticulum, producing either diffuse—or, more frequently—localized peritonitis with formation of an abscess. The anatomic changes produced by the more or less chronic form of the disease, in which infiltration of the wall of the intestine around the diverticulum is the most striking

feature, are of greater differential diagnostic interest. This inflammatory infiltration—which may involve all the layers of the wall below the mucosa—may recede by resorption of the inflammatory elements, or it may result in secondary fibrosis leading to the production of a firm mass within the wall of the intestine beneath the mucosa. Such thickening of the wall up to two inches has been described. The adjacent mesentery may be involved in the inflammatory and fibrotic process, thereby adding to the size of the mass.

Fig. 4-*B*. Examination eight days later shows the obstruction to be incomplete. Multiple diverticula are demonstrable. There is swelling of the mucosa of the sigmoid with localized narrowing. Clinical data: 45-year-old woman; history of crampy pain and diarrhea without blood for the last two weeks. Tender mass in the left lower abdomen, which had decreased in size at the time of the second examination.

The early inflammatory changes of diverticulitis show a more or less spastic portion of the intestine with wide transversely arranged folds producing a sawtooth or an accordion-like appearance, as first described by George and Leonard. This picture may simulate local inflammatory processes other than diverticulitis and can be produced also probably by destruction of the draining mesenteric lymphatics (Berg, Fleischner), but it cannot be confused with intrinsic cancer.

Abscesses produced by perforation into a walled-off area may be seen outside the bowel, associated usually with spasticity and other signs of diverticulitis. It is, however, important to state that the irritation may completely disappear, although a large inflammatory mass, with abscess formation, may still be present. If in such a case the barium does not enter the abscess cavity, an almost normal picture of the gut may result.

Increasing inflammatory swelling produces cushion-like projections, separating long-necked diverticula from each other. This picture has been described and illustrated extensively in the literature (Case, Spriggs and Marxer, and others). The process results in a bizarre, fringed contour, which, in spite of its marked degree, gives a deformity so characteristic that it cannot be confused with intrinsic cancer.

Differential diagnostic difficulties arise if inflammatory masses are arranged with fair evenness throughout the wall of the

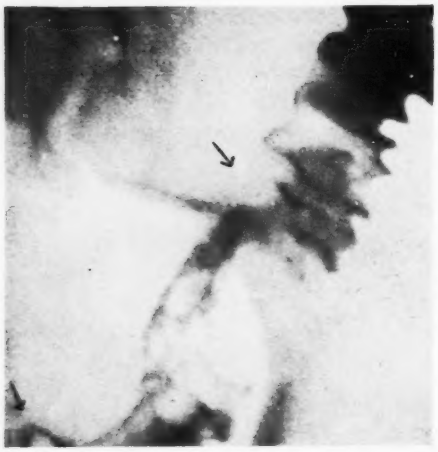


Fig. 5. Mucosal picture of a small area of cancer at the junction of the descending colon and sigmoid. Complete destruction of the mucosal relief within the area of cancer (*see arrows*). Swelling of the mucosal folds proximal to the cancer.

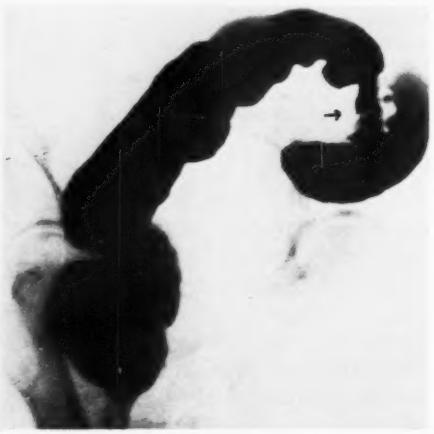


Fig. 6. Localized narrowing of the sigmoid (*see arrow*) in a patient with diverticulosis of the descending colon. (Cf. Figures 7 and 8.)

intestine beneath the mucosal surface (Fig. 1). The resulting concentric constriction usually has cone-shaped ends and extends in most cases over a fairly large part of the intestine. The wall of the intestine in the region of the cone-shaped beginning and ending of the lesion is usually not absolutely rigid, as can be seen by the changing lumen during any one examination. The constricted area itself may or may not change in size, depending on the degree of rigidity. The mucosal folds are preserved and may or may not be swollen; the size of the lumen in the constricted area itself may vary, from week to week, depending on the amount of cellular and serous, or fibrous elements.

The mimicry of cancer may, however, be still more marked, as the following case shows. A man, 55 years old, entered the hospital complaining of loss of weight and of rumbling gas in the abdomen. A questionable mass was felt in the left lower quadrant. The clinical diagnosis was carcinoma of the sigmoid. The barium enema showed a localized narrowing about two centimeters in length (Fig. 2). The narrowed area was not cone-shaped, but had a shelf-like margin on both ends, an appearance usually considered to be characteristic of the elevated edges of an intrinsic tumor. The area was completely rigid, and had not changed at a second examination done two days later. It did not relax with amyl nitrite. Proximal to this area there were a number of diverticula as well as definite spasticity of the gut. After inhalation of amyl nitrite this spastic area relaxed and the absolutely rigid region distal to it became even more striking. The rugæ in the suspicious area were, however, definitely preserved. In other words, the patient had, in addition to characteristic diverticulitis, a lesion which had all the signs of an intrinsic mass, such as absolute persistency in size and appearance. It had sharply defined edges and it was small in size, but the mucosa was not destroyed. The lesion, therefore, had to be localized within the wall of the bowel, but beneath the mucosa, and the diagnosis of diverticulitis without cancer was made. The resected specimen showed the narrowed area to be produced by fibrous masses within the wall of the intestine covered by normal mucosa. The roentgen-ray appearance of this lesion differed from that of a small cancer only by the appearance of the mucosa.

Another similar case in which the edge was not quite as sharp as in the preceding

case illustrates the same problem (Fig. 3-*A*). The lesion involved predominantly one side of the sigmoid. Three months later, the characteristic diverticulitis proximal and distal to the lesion had lost its inflammatory character—draining of a peridiverticular abscess had preceded the first examination by thirteen days—while the small fibrostenotic area was essentially unchanged (Fig. 3-*B*). It is characteristic of this type of lesion that, in the presence of localized rigidity and of all the evidence of a lesion within the wall of the bowel, the mucosal folds are preserved, although these folds may have a rather fixed arrangement such as is unusual for the constantly changing pattern of the normal colon.

The most difficult problem in differential diagnosis arises in cases with complete obstruction. It is obvious that it is difficult to diagnose a lesion when it is not outlined by barium. Case described, many years ago, the demonstration of the inflammatory mass in diverticulitis by the presence of an unusually long distance between the small intestinal loops and the brim of the pelvis on the left side. In cases of obstruction this mass may be clearly outlined by the air-filled small intestinal loops. It does not, of course, enable one to make a definite differentiation between inflammatory and cancerous mass. Usually all our efforts have to be concentrated on an attempt to bring at least traces of barium beyond the obstruction. The inhalation of amyl nitrite may occasionally be helpful. It is of greater importance to wait patiently for a few drops to pass through the stenosed area without over-distending the rectum, and to replace constantly any barium which may be emptied through the relaxed sphincter.

Occasionally films made after evacuation show barium beyond the point of obstruction, which was apparently transported upward during defecation. Sometimes *careful* inflation of the colon with small amounts of air (Fischer, Weber) may outline the stenosed area. If all these efforts are in vain, the appearance of the colon at the point of obstruction is often helpful. If a cancer is the cause of the obstruction, one will usually be able to see the surface

Fig. 7. The same patient as shown in Figures 6 and 8, showing the extensive diverticulosis of the descending colon. The area of narrowing is less distinctly visible in this projection (*see arrows*). A loop of small intestine is partially superimposed over the sigmoid.

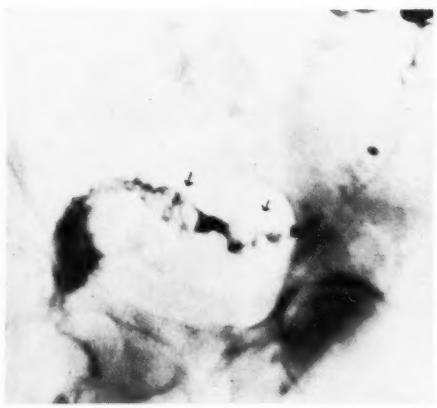
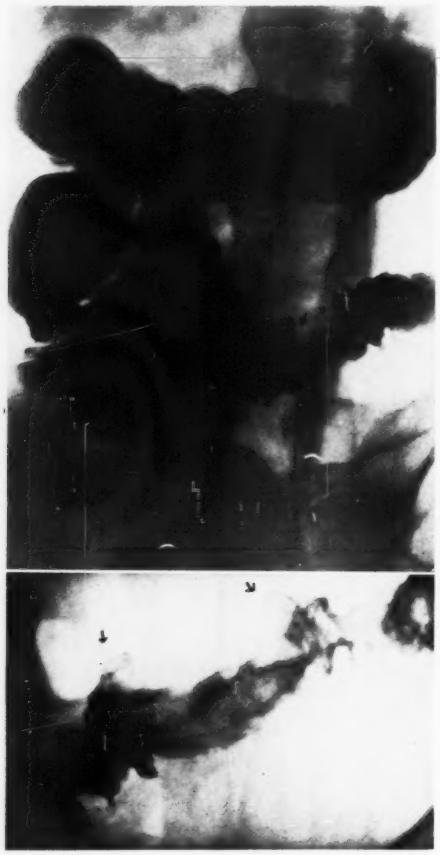


Fig. 8. The same patient as shown in Figures 6 and 7. Post-evacuation film shows definite destruction and ulceration in the area of the narrowing (*see arrows*). This patient had diverticulosis of the descending colon and cancer of the sigmoid Operation.

of the obstructing tumor, particularly during fluoroscopy or on films made after evacuation. In diverticulitis no definite tumor surface is visible at the point of obstruction; diverticula may or may not be demonstrated (Fig. 4). The diagnosis in such a case will usually be obstruction without demonstrable tumor mass. This is a diagnosis which is by no means pathognomonic of diverticulitis but which is very much against the presence of an intrinsic tumor. If a positive diagnosis is absolutely impossible, re-examination after one week of bed rest and bland diet may be helpful. If such a delay is not justifiable from the clinical picture or if the second examination again shows complete obstruction, a simple cecostomy without any attempt to explore the sigmoid may be the treatment of choice. It will relieve the obstruction. Another barium enema, one or two weeks later, very frequently will show the obstruction to be incomplete and will then enable one to recognize the pathologic process. This procedure will cause no loss of time if the lesion should turn out to be cancer, inasmuch as colostomy usually represents the first part of a two-stage resection of a sigmoid cancer, and it is, at times, good treatment for a stenosing diverticulitis.



Figs. 9-*A* and 9-*B*. *A*, Localized narrowing of the sigmoid (*see arrows*) in a patient with diverticulitis. *B*, Spot film shows destruction of the mucosa with ulceration in the area of the narrowing (*see arrows*) as evidence of cancer. Definite diverticulitis proximal and distal to the small cancer. Operation.

Fig. 10. Intramural extramucosal recurrence of cancer in the area of resection of a cancer of the lower descending colon. At operation a mass was found involving the wall of the bowel. The mucosa showed slight wrinkling, but it was not involved. Note the characteristic appearance of an intrinsic lesion with preserved mucosal folds.

In cancer of the colon the defect produced by the tumor shows sharply defined margins. Instead of the normal mucosal folds one sees the irregular tumor surface, usually with ulceration (Fig. 5). Very commonly the colon proximal and distal to the cancer appears normal. Spasticity and swelling of the mucosal folds may, however, be present, particularly proximal to the lesion as in diverticulitis. Diverticula may be present proximal and distal to the tumor and may complicate the diagnosis, particularly in small lesions. Figures 6 to 8 show the colon of a patient who had been treated for years for constipation. The examination showed multiple diverticula in the colon, with localized narrowing in the sigmoid. There was definite loss of the mucosal pattern with evidence of ulceration in this region, and the diagnosis of cancer with diverticulitis was, therefore, made. At operation the tumor was found to be inoperable due to liver metastases.

Not only simple diverticulosis but also definite diverticulitis may be present, together with a small cancer (Fig. 9). In other words, the presence of diverticula or diverticulitis does not help in the differential diagnosis between cancer and diverticulitis in a questionable, localized, narrowed area. One should hesitate to diagnose diverticulitis in the absence of demonstrable diverticula, but their presence does not help one way or the other.

In the description of the roentgenologic appearance of diverticulitis, I have stressed the importance of the recognition of localized fibrotic masses in the wall of the intestine. It is obvious that any intramural extramucosal tumor will produce a similar picture. A large variety of tumors may metastasize around the sigmoid and into its wall. Ovarian carcinoma and endometriosis do so most commonly. The differentiation of these from diverticulitis and primary carcinoma of the sigmoid goes beyond the scope of this paper and shall, therefore, be discussed only briefly. If such a tumor has destroyed the mucosa, a differentiation from primary carcinoma of the sigmoid is usually impossible. If it surrounds the intestine without involving its wall, it produces a pressure defect in an otherwise normal colon, thereby differentiating it from intrinsic carcinoma and from the majority of cases of diverticulitis. The localized intramural extramucosal tumor nodule may be differentiated from a similar diverticulitic lesion by its shape and by the absence of diverticula.

A roentgenologic differential diagnosis between extrinsic tumor masses and diverticulitis may become impossible if the masses have produced obstruction and incidental diverticula are present. Fortunately enough, however, it is usually much easier for the clinician to differentiate these lesions from diverticulitis, judging by his clinical examination and by the findings at operation. A pre-operative roentgenologic diagnosis is, therefore, not so badly needed as in the differential diagnosis between diverticulitis and intrinsic carcinoma of the colon.

If a resected cancer of the colon recurs locally in the wall of the intestine beneath the mucosal surface, it may also produce a picture of an intramural extramucosal lesion (Fig. 10). The history of such a case readily differentiates it from localized intramural inflammatory fibrosis.

SUMMARY

A differential diagnosis between cancer and diverticulitis of the colon is, in a fair percentage of cases, difficult or impossible during operation. The treatment of choice is different in most cases and a pre-operative diagnosis is, therefore, necessary. The roentgenologic differential diagnosis between the two diseases is easy in most cases, difficult in some, and impossible in a few.

Particular stress is laid on the differential diagnosis of localized fibrostenotic diverticulitis from cancer. In some of these cases, the characteristic picture of an intramural extramucosal lesion results. In such cases, the preservation of the mucosal folds is the only finding which differs from the appearance seen in a carcino-

matous lesion. It is possible that cancer produces occasionally a slightly fold-like picture. Usually, a differentiation of such an area from normal rugæ is not difficult. A warning against the indiscriminate interpretation of the rugal pattern seems necessary, however, particularly in cases in which the diagnosis rests exclusively on such interpretation.

The presence of diverticulosis or diverticulitis proximal or distal to a questionable narrowed area of the colon is of little or no value in differentiating cancer from diverticulitis.

Diverticulitis without demonstrable diverticula is rare.

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DISCUSSION

James T. Case, M.D. (Chicago): We have heard and seen a detailed presentation which seems to be almost perfectly complete, for which the author deserves commendation. The problem is a very real one and, even though he has shown a collection of instructive cases, I still feel that there will be many cases in which the diagnosis will be up in the air, after trying all of these devices of examination, and time alone will prove the lesion not cancerous.

The mucosal demonstration is a valuable means of differentiation and, as Dr. Schatzki has emphasized, if one decides upon the introduction of air to aid that purpose, it had best be done with extreme care. I notice that in one of his cases the colon was apparently over-distended with air. I would warn against using over-distention of the colon in doing air-enema combined studies. I have seen two cases of rupture of the sigmoid in the presence of diverticulitis, the rupture resulting from manipulation of the sigmoid and distention with a relatively small amount of air.

The speaker's recital of a case of a single colonic diverticulum in a patient who had had a pelvic operation, the tumor being found at the time of the operation, parallels a case of my own in which removal of a pelvic tumor was undertaken without preliminary x-ray study. At operation there was found also a tumor of the sigmoid, which was removed because it looked so surely cancerous; afterward it was found to be a diverticulitis with a single diverticulum.

I would like to report [the speaker showed slides] several cases to illustrate my remarks; not to show any difference of opinion with Dr. Schatzki but perhaps to emphasize what he has said.

My first is a typical case of carcinoma of the sigmoid in the presence of diverticula. This shows definitely the clean-cut, shelf-like defect of which Dr. Schatzki spoke—a clean-cut shelf at the distal end of the tumor, as shown by the barium enema, a finding always suggestive of cancer.

The next patient had a tumor of the sigmoid involving the bladder, which necessitated a resection of the fundus of the bladder along with the tumor. It was definitely carcinoma but in these films we see a diverticulosis of the sigmoid rather than a diverticulitis—perhaps the differentiation is more definite than in the presence of diverticulitis.

Next is the case of a dentist whose barium enema gave the findings of an irregular filling defect in the lower sigmoid, the differential diagnosis between carcinoma and diverticulitis not being easy. In the immediate films the appearance was such as to indicate carcinoma but in films made a few hours later, several small dots of barium developed near the lesion and gave the clue that we dealt with diverticula. Even at operation the surgeon could not decide, and he interrupted his surgery long enough to call me up for my final opinion as to cancer or diverticulitis. I voted for the latter and only a colostomy was done. Now, seven years later, the patient continues his dental work, but he has developed an ileocolic fistula, short-circuiting his inguinal colostomy and the stricture.

Ever since our earliest work in the colon we have emphasized the importance of making films of the sigmoid in the oblique projection, and, in cases of suspected diverticula, of making delayed films to allow time for the opaque material to work into the diverticula.

This case is a little more difficult—a man of 65 with a palpable mass in the left side of the abdomen and a very high grade of obstruction. I show you two sets of films—one without air injection, others with a little air carefully given. Because the mucosal folds were preserved throughout the lesion and because the delayed films showed penetration of barium into the diverticula, we were able to recognize the diverticulous nature of the lesion. Three years have passed under non-operative management and, although a certain grade of obstruction persists, by dietary and other means he manages to get along.

Whenever there is a question of diverticula, I always feel it is necessary to follow up the barium enema study by a study a few hours later or the next day to make sure we are not overlooking the diverticula.

MAURICE FELDMAN, M.D. (Baltimore, Md.): The diagnosis of a double lesion is always a perplexing one when it comes to pathology in the colon. In a differential diagnosis between carcinoma and diverticulitis, there is considerable difficulty and in some cases, of course, as Dr. Schatzki has pointed out, the diagnosis cannot be made with certainty.

In all these cases it must be borne in mind that the two conditions may coexist. The coexistence of carcinoma and diverticulosis occurs in a small percentage of cases. It seems to me that the mucosal relief film is the most important method of eliminating the two conditions. Even here one may find great difficulty. It is well known that in diverticulitis the size of the tumor has a tendency to change,

and this changing of the size of the tumor is a very important clinical fact.

Another important fact is that in diverticulitis the obstruction is rarely complete. It has been my experience that the law of averages is against the double lesion in those cases which show a diverticulosis, and in cases in which we find a localized lesion in the sigmoid, the law of averages would indicate the possibility of a diverticulitis although in a small percentage of cases this law is at variance.

Clinically, the picture is somewhat different, too. The mass is sausage-shaped and sometimes one may feel the mass while at other times it may not be felt at all.

Blood in the stools is ordinarily not seen in diverticulitis unless there is some complication such as ulcer or polypi. The presence of blood is variously reported as between 3 and 25 per cent of cases. In our experience we have seen the presence of blood in only a very small per cent.

DR. SCHATZKI (*closing*): I want to thank Dr. Case and Dr. Feldman for their discussions. It was a great honor for me to find that a man with the experience of Dr. Case did not disagree too much with me.

There are two points which I thought I had to discuss again when I saw Dr. Case's slides. One, in regard to the shelf. I do not wish to be misunderstood. The shelf is by all means much more common in carcinoma than it is in diverticulitis, and if I should see a shelf, particularly on both sides of the lesion, I would hesitate very long before all the other factors which, in the examination of a given case, are against cancer would make me exclude cancer.

Another thing about which I do not want to produce a misunderstanding is the importance of the demonstration of diverticula. The presence of diverticula does not help very much in the differential diagnosis between the two diseases but the absence does, and, therefore, it is so extremely important to prove the absence. That is often difficult. Therefore, we have to be careful before we make the statement that diverticula are not present.

The presence of diverticula may become important if they are present within the narrowed area because I think it is rare—I do not know if it occurs at all—that we find them right within a narrowed cancerous area.

THE HEMATOLOGIC DIAGNOSIS AND ROENTGENOLOGIC TREATMENT OF MYELOGENOUS LEUKEMIA[1]

By WALTER C. POPP, M.D., Section on Therapeutic Radiology, *and* CHARLES H WATKINS, M.D., Division of Medicine, The Mayo Clinic, *Rochester, Minnesota*

THE importance of an accurate hematologic diagnosis in myelogenous leukemia, particularly in relationship to the phase of the leukemia, cannot be overemphasized. The relative activity of any variety of leukemia is and should be of vital concern to the roentgenologist. For that reason we thought it worth while to present this subject from the viewpoint of the hematologist as well as that of the roentgenologist. We will present the hematologic pictures of four different phases of myelogenous leukemia and make suggestions as to both roentgenologic treatment and a method of follow-up examination which we think permits better control of a patient who has leukemia than is offered by sporadic treatment.

Myelogenous leukemia may be classified into four general types based on clinical and hemacytologic criteria. Diagnosis of acute leukemia may be made without difficulty. The disease usually has a rapid onset associated with fever, anemia, usually hemorrhage from the mucous membranes, petechiæ and purpuric manifestations in the skin, and, usually, a progressive enlargement of the spleen. The hemorrhagic phenomena may be so outstanding as to suggest a primary hemorrhagic disease, particularly purpura hemorrhagica. If the type of leukemia is aleukemic or leukopenic, it may be confused with acute aplastic anemia. Purpura hemorrhagica is usually readily distinguishable by the well known tendency of this disease to occur in remissions and exacerbations, in a relatively mild degree, and frequently over a relatively long period before an acute exacerbation occurs. Regeneration of erythrocytes is usually good and very little, if any, immaturity of the myeloid leukocytes will be found in the stained blood smear. Aplastic anemia frequently may be distinguished from acute leukemia by the absence of a palpable spleen. Examination of the stained smear reveals decreased regenerative activity of the myeloid leukocytes, rarely ever any slightly immature forms, relative lymphocytosis, thrombocytopenia, and a decreased number of reticulated erythrocytes. Acute myelogenous leukemia usually presents an elevated leukocyte count, but even if the leukocyte count is normal or if a leukopenia is present, examination of a stained blood film will establish the diagnosis. In acute myelogenous leukemia the predominating leukocyte is the myeloblast or stem cell, together with slightly more mature stages in the development of the myeloid leukocytes, leukoblasts, and promyelocytes. In any case in which this marked degree of immaturity associated with the clinical features previously listed is presented, the diagnosis of acute leukemia is warranted. In our experience treatment of this condition has been of no avail.

Subacute myelogenous leukemia usually is less dramatic in onset than acute leukemia, although, as the disease progresses, hemorrhage may occur as the result of reduction in the number of blood platelets in the peripheral circulation. Fever and anemia are usually associated with hemorrhage. The spleen in most cases is palpable, and examination of the stained blood film shows the predominant cell to be the leukoblast, with relatively few myeloblasts or stem cells, numerous promyelocytes and occasional myelocytes in more mature forms. It is most important to establish the diagnosis of subacute myelogenous leukemia for, in most instances, we feel that treatment is contra-indicated and it

[1] Read before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

is rather probable that most instances of subacute leukemia are really mild forms of the acute process, which means that roentgen therapy should be avoided. On the other hand, an occasional instance of the condition may present a rather high proportion of immature cells, associated with an almost equal number of relatively mature leukocytes, and a moderate reduction in blood platelets. In spite of the rather rapid onset of the disease, it will respond rather well to small doses of roentgen rays.

Chronic myelogenous leukemia usually is characterized by a gradual onset of anemia, a slowly progressive increase in myeloid leukocytes, and an insidious enlargement of the spleen. Many times the initial complaint of the patient is that of a tumor in the left side of the upper portion of the abdomen. For this reason it is often difficult to determine how long the disease has been present in a given instance. Examination of the stained smear discloses a marked increase in the number of myeloid leukocytes, the predominant cell being the mature neutrophil; metamyelocytes and myelocytes will be rather numerous. Cells that are more immature will be present but, when they are compared with the late phases in the development of myeloid leukocytes, myeloblasts, leukoblasts, and promyelocytes they will not be numerous.

The leukopenic or aleukemic phase of myelogenous leukemia may occur in any of the types of leukemia previously described, the final diagnosis resting upon a detailed study of the stained blood film. Because of the low leukocyte count, roentgen therapy must be administered with great caution and under no circumstances should patients who have the acute or subacute types of the disease receive roentgen therapy.

A number of methods of treatment for myelogenous leukemia are in use to-day, the more popular being irradiation of the spleen, long bones, mediastinum, and, more recently, the use of small amounts of radiation to the trunk at a relatively long target-skin distance, the so-called x-ray bath. We employ the method of treating the spleen, using a definite number of fields which, as a rule, are relatively small, the dosage being varied according to the phase of myelogenous leukemia under treatment. Our experience, based on about 350 cases, has led us to favor the use of moderate voltage (135 kv.) technics over all others we have tried.

The area of the spleen is divided into nine fields of approximately equal dimensions and of such size as to cover the entire spleen. There are four anterior, one left lateral, and four posterior fields. The four posterior fields correspond with the levels of the four anterior fields. These fields are numbered from one to nine, a procedure which provides nine fields of exposure over the spleen, thus permitting as many as nine treatments should that many be indicated, without repetition of exposure to any portion of the skin in a single course of treatment. Before treatment, a complete hematologic study is made of the erythrocytes as well as the cytologic aspects of the leukocytes. Should severe anemia be present, a transfusion of blood may be given. If only moderate anemia is present, transfusion is not indicated, since the majority of patients who have moderate anemia have a relative increase in erythrocytes as the leukocytes decrease in number. After the phase of myelogenous leukemia has been determined, the course of treatment is begun, and should first be directed to one of the fields of treatment near the lower edge of the spleen. On the following day a leukocyte count is made and treatment is continued, provided there has been no radical decrease in the total count. This method is followed daily until the leukocyte count reaches what seems to the hematologist to be a satisfactory level. This level, of course, varies considerably with the phase of the leukemia under treatment. For example, if the treatment is being carried out for chronic myelogenous leukemia, the number of leukocytes can be reduced more quickly than would be possible if a subacute or

aleukemic leukemia were present. If the count seems to have decreased too radically, treatment should be interrupted for from 24 to 48 hours so that the significance of such a decrease can be evaluated. We believe that a course of treatment should be carried out in a relatively short period of time, so that the patient can be allowed to maintain himself with no treatment for as long as possible; treatment is resumed when the leukocyte count begins to show a definite and persistent increase, particularly in the immature cells.

It is not necessarily true that the higher the count, the more treatment is necessary. It has been our experience that patients having relatively low counts of from 75,000 to 100,000 cells need more treatment to produce the desired effect than do patients who have a count of from 250,000 to 300,000 cells. In treating patients who have myelogenous leukemia, the leukocyte count should not be lowered beyond certain flexible values depending, of course, on the original count. If, for example, the original count in the presence of chronic myelogenous leukemia, before roentgen treatment, is approximately 75,000 cells, the course of treatment should not be carried beyond a point at which the count reaches approximately 25,000. On the other hand, if the original count is approximately 250,000, treatment should not be carried beyond a point at which the count reaches 50,000 to 60,000 cells. The reason for this precaution is that, after the patient has completed a course of treatment, the leukocytes continue to decrease in number for as long as a month or more and, should the treatment be carried too far, a dangerous leukopenia may develop. We have found that leukocyte counts which have been reduced to about 50,000, after a period of three weeks, will usually reach a normal level. We feel that the general condition of patients is better if they have a slight leukocytosis rather than a leukopenia. With a course of treatment being administered in a relatively short period averaging seven to nine days, it is easier to control leukemia and such a course facilitates additional treatment when resumption of such treatment becomes necessary. The use of a large number of small fields, in addition to permitting a fairly large number of treatments to be administered with little or no dermal reaction, causes almost no discomfort to the patient. It is not at all uncommon for patients to receive daily treatments of this type without experiencing any systemic reactions whatever.

In the presence of acute myelogenous leukemia there seems to be little that can be done therapeutically except to give transfusions of blood and provide supportive measures, and our experience has been the same as that of other workers in that roentgen treatment for this condition is contra-indicated except with rare exceptions.

In treating patients who have subacute myelogenous leukemia, the plan of treatment using nine fields is adopted as suggested previously. The dosage per field should, in our experience, not exceed 75 to 80 r measured in air. This is a small amount of radiation, but it has been sufficient gradually to alter the leukocyte count characteristic of this type of myelogenous leukemia. Treatment is administered daily until a satisfactory result is obtained. The treatment should be interrupted when the leukocyte count has reached a level of approximately one-third the original count. For example, if the original count was 150,000 cells, the course of treatment should be interrupted when a value of not less than 50,000 has been reached. Because of the radiosensitivity of the cells present in this type of myelogenous leukemia, there is great risk in using stronger doses because destruction of the leukocytes may get out of control, with serious consequences.

Chronic myelogenous leukemia is probably the least difficult form of leukemia to treat. The general condition of patients who have this type is usually better than that of patients who have the other types of myelogenous leukemia.

Roentgenologic treatment using nine

fields is arranged as previously suggested. The dosage employed is approximately 225 r per field. Daily sessions of treatment are employed so long as the count does not decrease radically. When the number of leukocytes reaches the desired level treatment is interrupted and the patient is allowed to go without treatment until the count shows a definite tendency to increase. The level to which the count should be allowed to fall is variable. The original level of the count is important in determining the level to which it should be reduced. For example, if the original count before treatment was 300,000, treatment should be stopped when it has reached a level of from 50,000 to 75,000. If the original count was 150,000, treatment should be carried on until the count has been reduced to a level of approximately 25,000 to 35,000. If the original count was 75,000, it can safely be reduced to a level of approximately 20,000 to 25,000. In the presence of this type of leukemia the leukocyte count should not be reduced to less than 20,000 unless the original count was not more than 20,000. With an original count of about 20,000, treatment may be administered until a level of about 10,000 is reached.

In the treatment of a patient who has aleukemic myelogenous leukemia a somewhat different problem faces the roentgenologist. Such a patient has a spleen which varies in size from mere palpable enlargement to a size sufficient to fill the left upper quadrant of the abdomen. The leukocyte count has variations from leukopenia of 2,000 cells to leukocytosis of 10,000 cells. In this type of myelogenous leukemia, treatment must be carried on with small doses and small areas of exposure. The daily dosage should not exceed 75 r. This amount of radiation usually causes little change in the leukocyte count but gradually causes a diminution in the size of the spleen and the patient's general condition improves. Usually, not more than five such treatments are necessary. Treatment is interrupted when the leukocyte count shows a tendency to decrease. For example, if the original count was only 5,000, treatment should be stopped when it has reached a level of approximately 4,000. If the count decreases after the first treatment, it is usually well to wait for from 24 to 48 hours before initiating the second treatment, because the count usually returns to the original level (or slightly higher), at which time treatment can be continued. If the original count was around 10,000, treatment can be continued until the count has reached a level of about 7,000. In treating patients who have leukemia of this type, we have seen remissions of the disease with almost normal health of the patient for as long as a year. Hematologic studies and palpation of the spleen should be carried out monthly.

When a course of treatment for a patient who has any type of myelogenous leukemia has been completed, a monthly leukocyte count should be taken and, provided the leukocytes do not tend to increase rapidly in number and immaturity, treatment should be withheld until such time as a definite increase manifests itself. Rather than to allow the leukocyte count to reach a hazardously high level, treatment should be resumed whenever a tendency to progressive increase becomes apparent. This procedure permits better control of the leukemia and the patient may continue to be a useful individual with the least amount of incapacitation. Moreover, less treatment is required to bring the leukemia under control.

The follow-up procedure for this condition is almost as important as the treatment itself. With such a varying clinical entity as myelogenous leukemia, as in the case of other types of lymphoblastoma, a statistical analysis of survival after treatment among patients suffering from this disease does not show a true picture of the value of roentgen therapy. It is the consensus, however, that, as a palliative measure, roentgen therapy surpasses all other methods of treatment.

The technics which we have outlined herein seem to be definitely superior to

other methods which we have tried. Three things should be avoided. First to be shunned is the not infrequent habit of some roentgenologists of giving "a little bit" of roentgenologic treatment to the spleen without a clear knowledge of the true status of the disease and without a definite plan of treatment. The second action to avoid is irradiation of the long bones of the average patient since, in our experience, such irradiation occasionally has produced a rapidly fatal outcome in cases in which the patients otherwise were progressing favorably. The third procedure to be avoided is the use of 200 kv., even in mild doses. In our experience this type of treatment may produce a more spectacular and more rapid immediate result than that which we have discussed, but the condition of patients so treated seems to become refractory much sooner than that of patients who have been treated with a technic similar to the one we have discussed.

SUMMARY AND CONCLUSIONS

This paper offers nothing new in the treatment of subacute, chronic, or aleukemic myelogenous leukemia, but it does offer a definite therapeutic plan which we have found to be very satisfactory. Treatment administered systematically, rather than sporadically, keeps patients in better general condition. We have also found that, by using a definite number of well defined roentgenologic fields at the time each course is given, the epidermis of the patient remains in much better condition and thus tolerates considerable repetition of treatment. We believe that by using a relatively small quantitative dose to each field of treatment, the leukocyte count decreases more slowly than it would otherwise and that the complete course can be administered without systemic reaction in almost every case. We have no evidence that stronger doses than those we have used produce better results in the average case.

BRIEF OBSERVATION ON THE ERYTHROCYTE SEDIMENTATION IN RELATION TO IRRADIATED MALIGNANT TUMORS[1]

By MANUEL F. MADRAZO, M.D., *Mexico City, Mexico*

IF we draw blood from an individual and mix it with an anti-coagulating solution, *e.g.*, citrate of sodium, placing it in a test tube, the combined elements will gradually form a sediment, the rate of sedimentation depending, apart from the technic followed, on the physiological variations of the individual, on the surrounding temperature, the hygrometric state, and on the atmospheric pressure. The measurement of the sedimentation rate, easily carried out, constitutes the test, but it is necessary to take into account the factors pointed out. It has been found that it is slower in men than in women, in whom it is influenced by the menstrual and premenstrual period and by pregnancy.

It is generally admitted that, by its coagulating properties, the fibrin of the plasma is an impediment to sedimentation. Hence, to study the phenomenon in question, it is necessary to counter-arrest the effect of the intracellular ferment which produces the passage of the fibrinogen of the blood into the solid state. This is obtained by means of different chemical substances, but in Westergren's technic, which we have followed, citrate of sodium is used. The majority of other technics are modifications of that of Westergren.

Several theories as to the cause of corpuscular sedimentation have been propounded. Stokes' Law was primarily stated as its only cause, but later studies show that the mechanism is much more complicated.

Not until Fahraeus presented his paper at the Surgical and Gynecological Congress in Stockholm, Dec. 1, 1917, was the erythrocyte sedimentation test first used in general clinical work and in some specialties. Known since old times, it had been explained by the humoral theory and has been the object of more or less interesting observations until the studies of Virchow in cellular pathology deviated the importance of the macroscopical study of blood. Subsequently, investigations of Biernacki, Müller, Hunter, Davy, and many others had not awakened the interest which took place with the work of Fahraeus, from which were derived a series of clinical experiments, among which stand out those of many investigators of different nations: Tsudumi, Sakal, Bonninger, Hermann, Linzenmeier, Kürten, Hober, and others, Cutler and Gram in the United States, and Forestier in France.

Fahraeus showed that in the phenomenon named after him a special grouping of red cells takes place, giving rise to various opinions, attributing it to flocculation of colloids, as Poppert and Kreindler believe, or to other factors, such as the stability of the electric charge, the blood viscosity, the superficial tension of albumens, etc. An interesting fact exists: at greater viscosity the rate of sedimentation increases, contrary to what happens with solution of albumen, which, being of low viscosity, delays sedimentation. This may be explained because these latter are formed of very small molecules and albumen has a high dispersion, giving rise to a larger stability of the blood plasma, while the increase of fibrinogen and of globulins, which have a low dispersion, raise the sedimentation rate when the blood viscosity diminishes.

There is an inverse relation between the amount of cholesterin and the sedimentation index. This relation has been studied by Linzenmeier and Kürten. Hober demonstrated the negativity of the electric charge of the normal erythrocytes. When that electric charge is neutralized, the sedimentation of red cells is hastened.

[1] Presented before the Fifth International Congress of Radiology in Chicago, September 13-17, 1937.

This point of view of the influence of the electric charge has been accepted by Fahraeus. Exposure to ultra-violet rays diminishes the potential of the electric charge of red cells and increases the rate of sedimentation, probably because it diminishes at the same time the repulse force between them, since they have always a negative electric charge. Likewise, theories have been established on the phenomenon we are studying, relating it to the index of blood calcium, to the CO_2, to the diminution in size of the red cells and their hemoglobin content, and to the alteration of the relation between albumen and globulins. Grandwohl attributes it to the *p*H, since acidosis delays and alkalosis increases the erythrocyte sedimentation rate.

Experiments of Maccabruni and Snaper tend to demonstrate that the corpuscles do not intervene in the sedimentation rate. If red cells of patients sick with some infectious disease are mixed with the serum of healthy individuals, sedimentation takes place normally. On the other hand, erythrocytes of healthy individuals mixed with diseased plasma, present an abnormality in sedimentation; from this these researchers deduce that sedimentation is influenced by the plasma and not by the corpuscles.

From what has been said, it is inferred that there are numerous factors influencing corpuscular sedimentation, and most probably there exist other causes still unknown.

The test is not specific, for which reason it has no diagnostic value, but, on the other hand, it does seem to have a great prognostic importance in some diseases. "The value of the reaction of corpuscular sedimentation is that of a general biological phenomenon, which always gives practically exact figures in normal individuals and may be raised in a passing or constant manner in the majority of acute or chronic diseases."

Cooper and Kürten mention acceleration in malignant tumors, believing this to be due to an increase in the amount of cholesterin in the blood. According to Forestier and Tonnet, it is very high in parenchymatous neoplasms, owing to phenomena of paradoxic albuminosis in the blood. Gram found a rise in the fibrin content of the plasma of nearly all infectious diseases, in carcinoma, etc.

The sedimentation test has come to be recommended as a help in differentiating benign from malignant tumors. It is generally admitted in the differential diagnosis of benign and malignant tumors that, under similar conditions, an increased sedimentation rate means malignancy. When not complicated by infection, fibromas, lipomas, and fibromyomas have no influence on sedimentation. However, in the earliest onset of a malignant tumor, the sedimentation rate may not be altered. It has been observed that there is no change in the phenomenon of Fahraeus in some cases of cancer of the cervix and of scirrhous carcinoma of the esophagus and stomach. Peters has pointed out that normal sedimentation figures are observed in epithelial cancer, as in that of the tongue and of the penis. Katz and Leffkowitz mention as possible causes affecting the stability of the formed elements, the anatomical character, its situation, vascularity, tendency to invade the neighboring tissues, degree of reabsorption of the tumor tissue, of the inflammatory reaction and the presence and state of metastasis. These authors think the reason why malignant tumors present modifications of sedimentation is because they show a tendency to disseminate.

I believe the study of the sedimentation rate to be of great interest in cancerous patients, following the evolution of the disease and relating it to irradiations. As pointed out, in the probable causes of sedimentation, many occur in patients with malignant neoplasms, either chemical modifications or biological elements constituting it. I ignore up to what degree irradiations modify the electric charge of the red cells, but if ultra-violet radiations are capable of altering it and producing acceleration of the sedimentation, it is

logical to suppose that x-rays in the form of deep therapy, as well as radium irradiation, have a more marked effect than ultra-violet rays. With such belief, I have irradiated guinea pigs and have invariably observed that the irradiation effect gives rise to an increase in the sedimentation rate. I chose these animals because they can be bled easily by puncturing the heart. At the same time, apart from the already well-known organic changes which irradiation causes when the animals survive to the irradiation, the sedimentation rate becomes normal, parallel with the descent of the irradiation curve. Consequently, I do not agree with the opinion somewhere expressed that irradiations do not affect the rate of sedimentation. I have measured the sedimentation rate in women suffering from uterine fibromas and found it normal before irradiation, and modified afterward, the same as happens with guinea pigs. The same thing occurs in cases of prostatic adenomas.

The cases studied are not in a sufficient number to allow me to present statistics and, still less, conclusions. The object of this paper has been to awaken among radiologists an interest in the study of a subject well worth while, in my opinion. Compared with other diseases, principally tuberculosis, I have found very few papers treating the modifications of the corpuscular sedimentation in cancer relating to irradiations, and in some of these papers contradictions are found. If investigations are carried out at the same time in different countries, following the same technic and a similar scheme in the observations, by the next International Congress we shall be in a position to confirm the observations made or to rectify them. As I have previously said, those that I have had the opportunity to follow make me foresee important possibilities in the study of the sedimentation rate with respect to the evolution of cancer in patients treated by irradiation.

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FATE OF CRANIAL DEFECTS SECONDARY TO FRACTURE AND SURGERY[1]

A FOLLOW-UP STUDY OF 150 PATIENTS

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STUDIES of the persistence of fracture lines have been much neglected in medical literature, though the scientific and medico-legal value is of considerable importance (Stewart, 1925; Glaser and Blaine, 1936). From a clinical viewpoint it is extremely valuable to be able to prognosticate the persistence of cranial defects, either traumatic or operative, and further, to be able to inform the patient of the length of time necessary for a bone graft to fill such defect.

An intensive and prolonged study shows that the healing of linear fractures depends, to a great degree, upon the age of the patient. The disappearance of the fracture line occurs much earlier in children than in adults. The width of the fracture also plays a part in the prolongation of its visibility upon the roentgenogram. Occasionally, if the fractures are too wide, absorption rather than healing of the fracture takes place. Ossification around the fracture and of the fracture itself may also occur. The location of a fracture in the skull also is an essential factor to be considered. In certain instances, fractures in the occipital area take much longer to disappear than in other parts of the cranium. It is well known that delayed union, and sometimes non-union, of fractures of the long bones occur: whether a similar bio-chemical process takes place in skull fractures is a matter yet to be ascertained.

In this series of 150 patients, x-ray films have been taken at varying intervals following fractures to determine the ability of the skull to repair such defects. In all instances, attention was directed to a duplication of the x-ray technic carried out in the original roentgenograms. The entire group has been classified as follows:

1. Linear Fractures:
    1. Disappearance of fracture line in children under six years of age.
    2. Disappearance of fracture line in adults.
    3. Widening of fracture line in children and adults.
2. Depressed Fractures:
    1. Without elevation of fragments.
    2. Hammering out of fragments.
    3. Cranial defects.
    4. Replacement of fragments at time of operation.
    5. Osteoperiosteal grafts.
3. Osteoplastic Bone Flap:
    1. Normal bone flap.
    2. Degeneration of the bone flap.

LINEAR FRACTURES

1. *Children.*—There are eight children under six years of age in this group. Fading of the fracture became apparent within two months after injury, and within six months after injury the fracture line disappeared in every instance, excepting one.

Case 1, S. F., male child, aged six years. This child was struck by an automobile, the wheels running over his head, giving him a tremendous fracture of the skull with many branches of the fracture line. There was considerable separation of one of the more extensive fractures, passing practically the whole length of the vault. The child was rendered unconscious for several hours, was irrational for several days, but after a stay of some three weeks in the hospital, made an uneventful recovery.

[1] Accepted for publication in September, 1939.

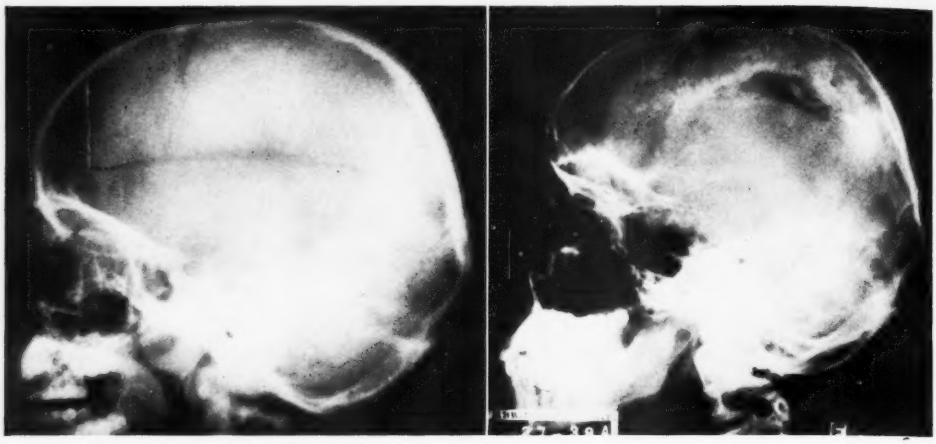

Fig. 1. Fig. 2.

Fig. 1. Lateral view of skull showing wide separation of fracture line. Note the ramifications.
Fig. 2. Lateral view of same case taken nine years later. The skull has become irregular around the fracture line, and there has been a marked absorption in the center of the fracture. Parts of the linear fracture have, however, disappeared.

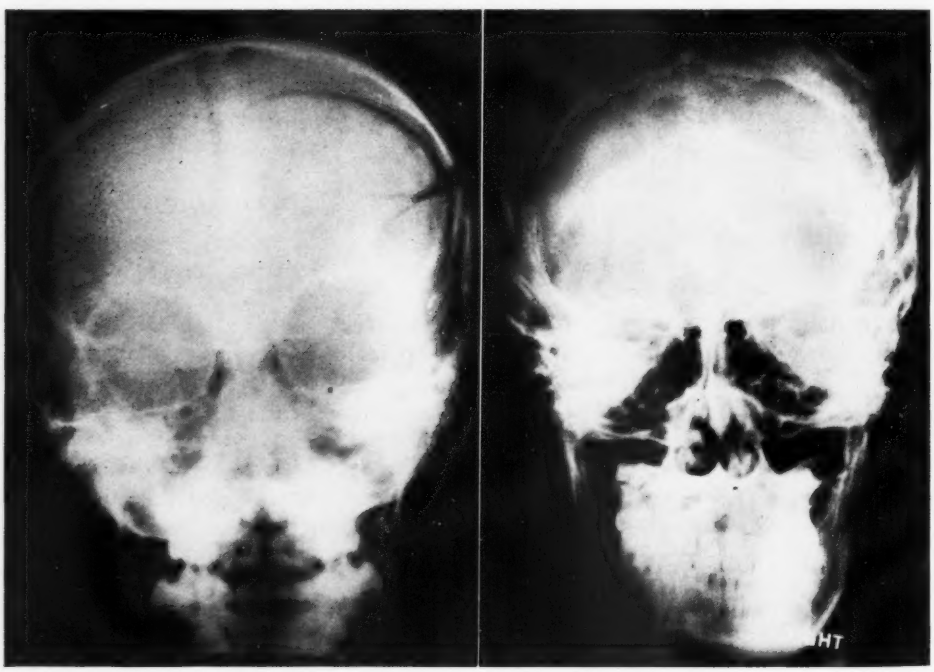

Fig. 3. Fig. 4.

Fig. 3. Same patient as shown in Figure 1. The fracture is of a stellate type with a very wide separation.
Fig. 4. Same patient nine years later. All the fracture lines have disappeared except the widely separated area. This has become even wider due to absorption around the fracture line. The patient is now 14 years of age.

The extent of this fracture may be noted in Figures 1 and 3. X-ray films were made over varying intervals of time, and finally after a nine-year period the lateral film revealed a wider absorption of the center of the linear fracture. There was increased calcification of the bone above this fracture, with definite calcification along both margins of the transverse branch of the main lateral ramification. In the postero-anterior view it may be noted (Figs. 2 and 4) that most of the fracture lines have entirely disappeared. However, the main central fracture instead of disappearing has become definitely widened, with considerable absorption of the area superior to the crevice. In this instance, the continuation of the defect was due to the extensiveness and wide separation of the fracture. In addition, the increased calcification about the fracture line, and superior to it, was a rather unusual occurrence in a child of this age, this being the only case in our series which presented this picture. In the remaining patients, the fractures disappeared within the six-month period. Without doubt this fracture will remain visible during the entire life of the patient.

2. *Adults.*—In the 55 cases of adult linear fractures, the disappearance time was extremely variable. The earliest time for fading, so that the fracture was not distinguishable unless the original films were reviewed, occurred within seven months. It is quite apparent that those fractures which existed in the occipital region took a much longer time to disappear than those appearing in other parts of the skull. In this group, some fractures of the occipital region were visible for an eight-year period. In four examples, instead of a healing, a widening of the fracture with absorption about the edges occurred. In all these cases, the separation was rather extensive, though in some, a wide separation did not follow such a course. The following five cases present examples of the varying kinds of fading, from a seven-month period to an eight-year period, and also give an example of absorption about the fracture.

Case 2, R. S., aged 40, a frontal parietal fracture faded to practical invisibility after seven months.

This man had a severe head injury on Sept. 1, 1935, when he was rendered unconscious, but did not have a skull fracture clinically or roentgenologically. Following this injury he developed a disabling post-concussion syndrome, associated with headaches and dizziness, as well as marked

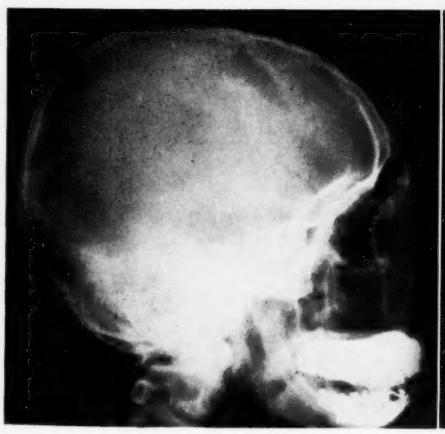

Fig. 5.

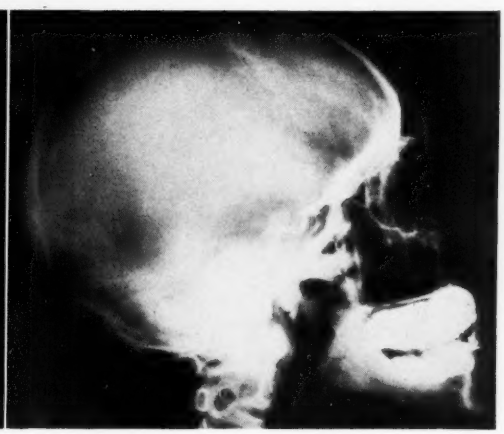

Fig. 6.

Fig. 5. Linear fracture in adult aged 40 is clearly visible in the frontal region.
Fig. 6. The same linear fracture has practically disappeared within seven months. Without the original films the existence of this fracture could not be definitely ascertained.

nervousness. At a later period, he developed a post-traumatic epilepsy. During one of these epileptic seizures, on Dec. 15, 1938, he fell to the ground. This time he did receive a skull fracture and was again rendered unconscious (Fig. 5).

His headaches and dizziness became more severe, and he tired more readily. His vision became blurred and his unconscious attacks increased in frequency so that he had as many as 30 attacks in one year. Physical examination revealed a well built, sturdy individual. His septum was deviated slightly to the right. There was a polyp in his right nostril, and his teeth were in relatively poor hygienic condition, with partial upper and lower plates. In addition, he had a moderate kyphosis. Neurologic examination revealed some tenderness in the right occipital region. There was an irregularity in his pupils, and he reacted sluggishly to light and distance. His reflexes were within normal limits, but there was a marked tremor of his eyelids. He had a tendency to sway while standing with his eyes closed, although this latter was not a true Romberg. His blood and urine were normal. Wassermann test was negative. X-ray examination, repeated on June 2, 1939, some seven months after the original injury, revealed a marked fading of this fracture, although a definite diagnosis could not be arrived at unless the original films were reviewed (Fig. 6).

Case 3, J. G., aged 34, weight 144 pounds, height 5 feet 3 inches. A large fronto-parietal fracture, fading to near invisibility 10 months after injury, and to absolute invisibility in 14 months after injury.

This man had a severe head injury on April 20, 1938, falling a distance of 12 feet to a concrete floor below. He was immediately rendered unconscious and remained so for one-half hour. He had no memory of events during the first 14 hours. In addition to his skull fracture,

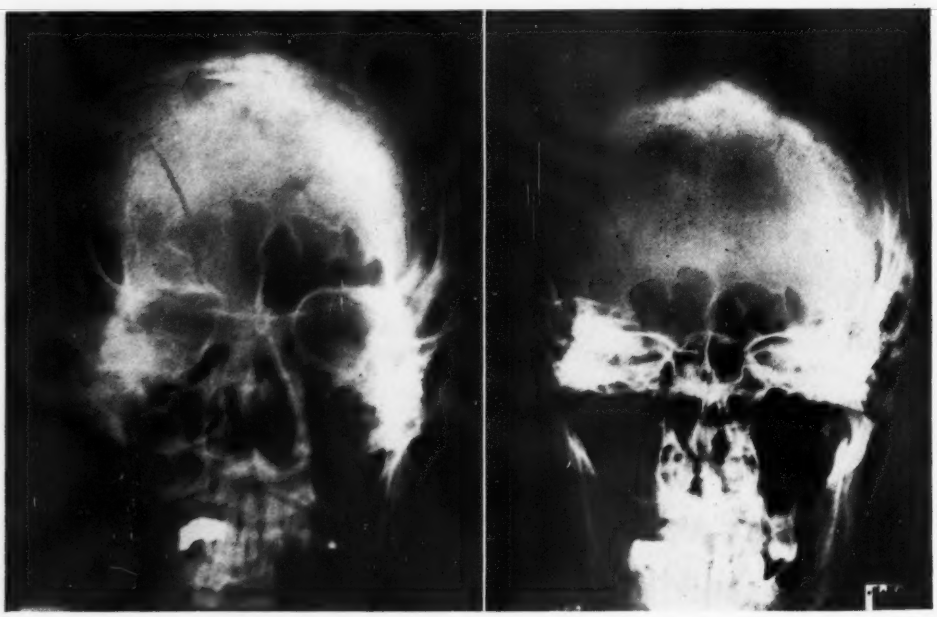

Fig. 7. Fig. 8.

Fig. 7. J. G., aged 34, received extensive frontoparietal temporal fracture. There was considerable separation in the frontal region.
Fig. 8. Film of the same patient, taken 10 months later. Note the gradual fading of the fracture line.

he had a fracture of the left radius and ulna at the wrist and developed a cerebral spinal rhinorrhea, as well as an associated diabetes insipidus, drinking between two and three gallons of water a day. His right eye was closed and swollen and he had numerous body abrasions and contusions, but his neurologic examination was essentially negative. After he regained consciousness, he complained of headache, but was entirely without dizziness. He remained in the hospital until May 15, then rested at home for an additional three weeks. He had few complaints and gradually returned to his occupation as storekeeper. His diabetes insipidus has persisted to the present time, though now there is only a slight excess in his water-drinking.

X-ray examination on April 21, 1938 (Fig. 7), showed a fracture in the right frontal region, about three inches in length, extending through the frontal sinus and involving both the anterior and posterior walls. The entire sinus was clouded and filled with blood. Another fracture was visible in the parietal region on the right side, about five inches in length, entering the mastoid area, which also was somewhat cloudy. X-ray film taken on Feb. 6, 1939, 10 months after injury (Fig. 8), revealed a remarkable fading of the fracture line, far more than would ordinarily be expected. The rather wide frontal fracture was narrowed and the parietal bone fracture was barely visible. X-ray films taken on June 29, 1939, 14 months later, revealed a nearly complete disappearance of the fracture line. The only indication discernible was the lower border of the parietal line where it had entered the mastoid. This in itself would not be recognized as a fracture, unless the previous films were used in comparison.

Case 4, A. S., aged 23, weight 170 pounds, height 6 feet one-half inch. An extensive fracture extending over the entire lateral half of the skull showed marked fading, though readily distinguishable upon x-ray examination, 17 months after injury.

On July 5, 1937, this young man was struck on the head by a timber from an oil derrick, and was rendered unconscious for a lengthy period of time. He has no recollection of events for a period of two weeks after the injury. He remained in the hospital seven weeks, and then reported for office examination. On Dec. 7, 1938, he still complained rather severely of headaches and dizziness, weakness, fatigue, irritability, and back pain. After

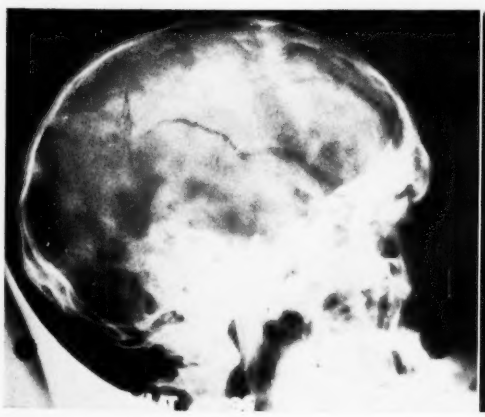

Fig. 9.

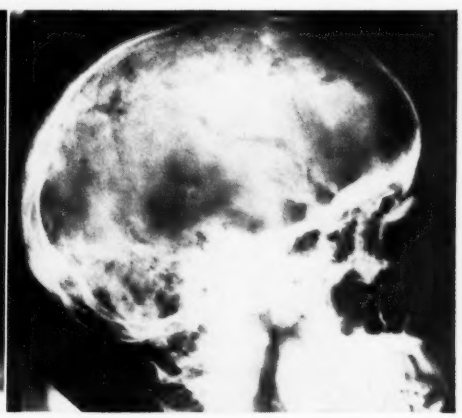

Fig. 10.

Fig. 9. A. S., male, aged 23. There is extensive fracture with wide separation over the lateral side of the skull.

Fig. 10. Same patient, 18 months later, still showing evidence of the fracture present. However, marked fading had occurred.

a careful observation of the patient, it was quite apparent he was exaggerating his complaints, and that his disability was not as great as pretended. A secret investigation was then carried out. This revealed that he was able to work, and that his complaints were merely accentuated for financial gain. Following a settlement of $300, purely on a nuisance basis, this man returned to work.

Physical examination revealed that his septum deviated to the right, his tonsils were large and infected, and he had moderate point tenderness over the lumbar sacral region. Neurologic examination revealed an area of a well healed superficial abrasion in the right temporal region, and a scar in the angle of the left infra-orbital region. He was unable to distinguish test odors; however, this was later proved to be feigned. Blood and urine were entirely negative. His Wassermann was negative. X-ray examination, on July 6, 1937 (Fig. 9), revealed a large fracture of the left lateral skull wall, extending the entire width of the skull. X-ray examination on Dec. 8, 1938 (Fig. 10), disclosed an extensive healing of this fracture.

Case 5, W. B., male, aged 41, weight 175 pounds, height 5 feet 9¾ inches, a policeman, was injured in a motorcycle accident. An extensive stellate fracture in the occipital region was still evident nine years after injury. This patient was immediately rendered unconscious and remained so for six hours. He recollects nothing for a period of three weeks after the injury. After a two and one-half months' leave of absence, he returned to work. He did not, however, co-operate well with his superiors, and developed a persecution complex; in various ways it was noted that he exhibited a distinct change in personality. Associated with his mental disturbances, he developed headaches of a severe degree, and some degree of nervousness. Nine months after injury he had his first unconscious attack. Initially these attacks occurred every three weeks, but in the past six years they have occurred on an average of twice a week.

Physical examination revealed a clouded right tympanic membrane. His dental hygiene was extremely poor. Neurologic examination was essentially negative, except for marked irritability and sluggish mentality. X-ray examination on March 20, 1930, revealed an extensive fracture in the posterior part of the right parietal bone, somewhat crescentic in shape, with a diastatic separation of the lambdoid suture. On the left side was seen a branching fracture, extending down into the left mastoid

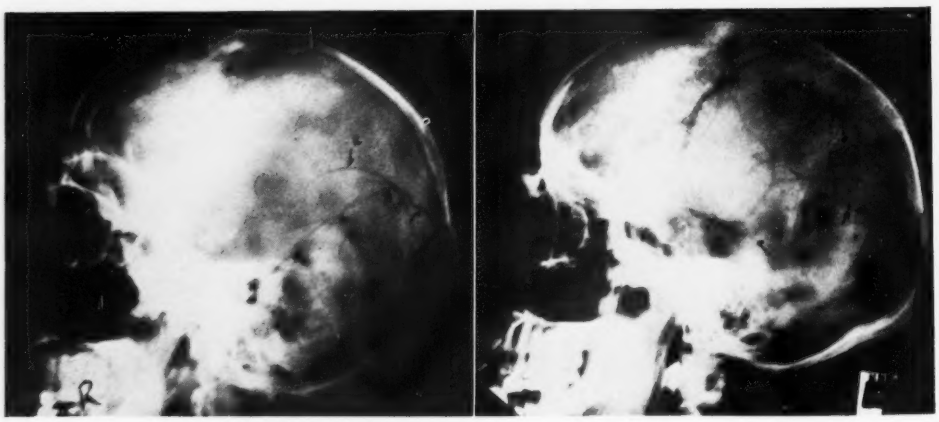

Fig. 11. Fig. 12.

Fig. 11. W. B., male, aged 41, had a fracture in the posterior part of the parietal and occipital bone.
Fig. 12. Films taken nine years later still reveal the evidence of fracture.

cell, as well as numerous crisscross ramifications (Fig. 11). In Figure 12 may be noted the remains of this fracture, still visible nine years after injury.

Case 6, E. M., male, 53 years of age, weight 184 pounds, height 5 feet 10 inches, had an extensive fracture in the area of the coronal suture, extending into the parietal and temporal bones. X-ray films taken 18 months after the injury showed a fading as well as a widening of the fracture lines.

On Nov. 12, 1935, while rolling some barley in a mill, he fell from an elevator, was immediately rendered unconscious, and had very little recollection of events for the ensuing several weeks. He remained in the hospital for a period of four months. In March, 1936, he returned to work, but was discharged because of inefficiency. He realized this inefficiency, and recognized that he could not do his former work as a foreman of the mill. He complained of headaches and dizziness, weakness, ringing in the ears, failing vision, and back pain. Physical examination on April 19, 1937, revealed a corneal opacity and a loss of vision in the right eye. There were some old scars, well healed, across his back. Neurologic examination showed some tenderness to pressure in the right temporal region, with a definite irregularity of the skull wherein elevations and depressions could be palpated. A large coronal irregularity felt as if the bone were completely absent. He was unable to distinguish test odors, but could detect taste tests. Due to his loss of smell, he was, however, unable to taste the finer, more selective food substances. He had a perfect realization of his inability to carry out work, and knew that he was haphazard, careless, and irresponsible.

X-ray examination on Nov. 14, 1935 (Fig. 13), showed an extensive fracture involving the frontal and parietal bones. X-ray examination on April 20, 1937, demonstrated a wide absorption of this fracture. The fracture on the left parietal bone, running parallel with the coronal sutures, measured 3 mm. in its greatest spread, while the upper and lower ends of the fracture were closely approximated—in places, separated by less than 1 mm. The fracture line in the right parietal bone, running parallel with and posterior to the coronal suture, reached a maximum spread of approximately 9 mm. in the lower part. The spread averaged about 5 mm. and at certain points bony bridging occurred from edge to edge.

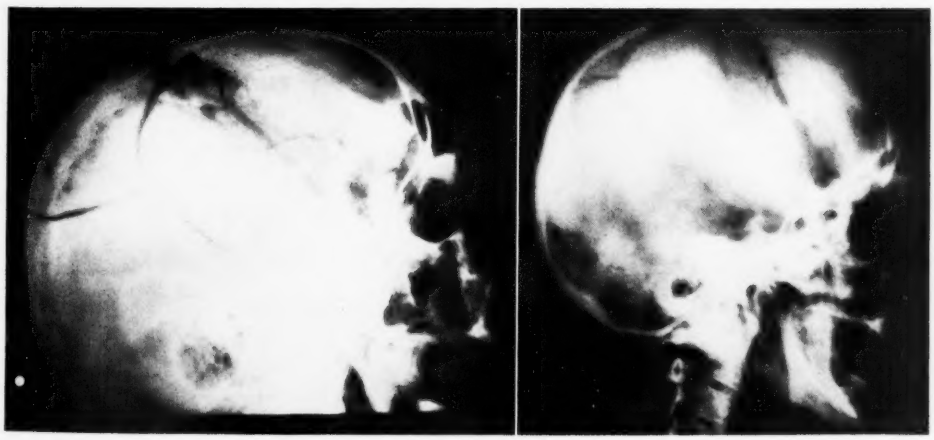

Fig. 13. Fig. 14.

Fig. 13. An extensive fracture of the skull with wide separation of the fragments.
Fig. 14. Film of same patient, taken 18 months later. There is a gradual fading of most of the fracture line. A large wide fracture in the area of the coronal suture has become even wider due to absorption about the edges of the fracture.

Additional fractures were seen running in different directions in the left parietal bone, also involving the squamous portion of the left temporal bone. Figure 14, taken one year later, showed very little change. In it, some depression is seen at the vertex. The anterior fragment has settled down a distance equal to the complete thickness of the skull wall.

DEPRESSED FRACTURES

There were 79 individual cases of depressed fractures. In the group without operative elevation, the fragments fused together to form a solid rounded posterior edge, as well as a similar rounded anterior edge in every instance. The time of fusion varied from 10 to 14 months. In one instance, a bone flap was turned down and the fragment hammered in place. With the bone flap replaced, no evidence of the fracture was visible after a 10 months' period. This operation was too formidable, and is deemed unwise for the treatment of depressed fractures. In those cases in which the fragments were removed and a defect in the skull remained, this defect never decreased in size. The only change noticeable was a rounding of the serrated edges within an eight months' period of time. This occurred in ages from six to sixty years. In certain instances the fragments were replaced. If spaces were left between the replaced fragments and the edges were not approximated, or bone dust not inserted between these edges, the x-ray evidence of separation is maintained permanently, up to five years. There is no indication of fill-in, yet, to palpation, the surface appeared as if solid bone were present. In order to maintain the position of the fragments in close approximation, a catgut screen has been devised. This assures that the smaller pieces of bone will not become misplaced, and the scalp may be firmly sewed together. In such instances asepsis must be assured and the dura must be intact. Graft or suture is necessary in all such cases. Experimental studies by Glaser and Thienes demonstrate that an animal membrane graft is just as efficient as fascia lata. It has been found

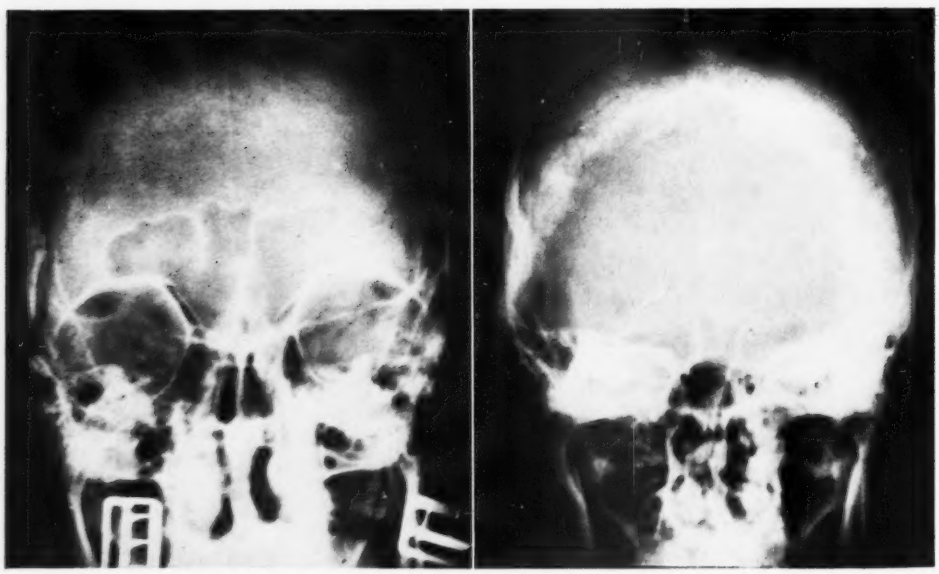

Fig. 15. Fig. 16.

Fig. 15. A depressed fracture in the temporal region which has not been elevated. Note the fragmentation of the depressed bone.

Fig. 16. A film taken 23 months later showed a fusion of the fragmented particles, with a rounding and smoothing of the dural edges.

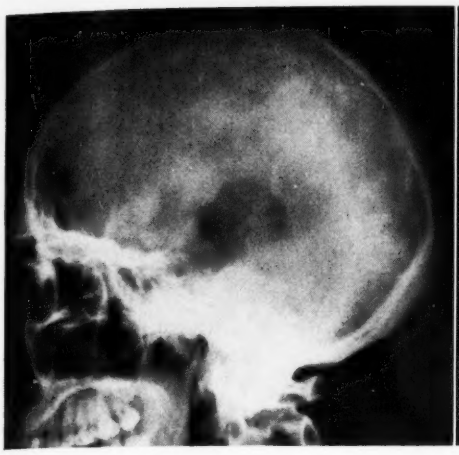

Fig. 17.

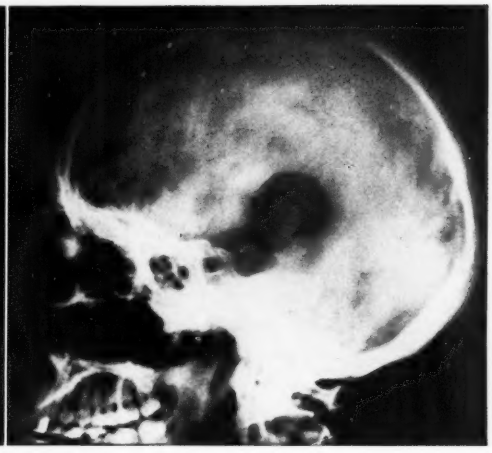

Fig. 18.

Fig. 17. A depressed fracture with elevation and removal of the fragments. Note the fine serrated edge.
Fig. 18. X-ray films taken eight months later revealed the smoothing of this serration, without any decrease in size of the defect. This defect never became smaller.

that osteoperiosteal grafts are the most satisfactory, both from a technical and a clinical standpoint (Glaser and Shafer).

Case 7, F. S., aged 57, weight 175 pounds, height 5 feet 10 inches, received a depressed fracture which was not elevated. X-ray films taken 23 months later showed a fusion of the fragments, with a rounding and smoothing of both edges.

This man fell a distance of 12 feet, striking the ground, being rendered unconscious for six minutes. Upon examination he was found to be entirely symptom-free, except that he was somewhat drowsy. He had a blood pressure of 165/105, and a definite auricular fibrillation. The hygienic condition of his mouth was extremely poor; tonsils large and infected. In addition, he had a fracture of the clavicle and two metacarpals of the left hand. Neurologic examination revealed an edematous and discolored face, rupture of the left tympanic membrane with hemorrhage, tenderness, and swelling over the temporal region of the scalp. Due to this man's auricular fibrillation and a complete absence of neurologic signs of a focal or generalized nature, elevation was deferred. He was discharged from the hospital at the end of four weeks and returned to his usual labor six weeks after injury entirely symptom-free. The auricular fibrillation persisted, and because of the absence of any head symptoms, elevation was never carried out. He died seven years later from heart disease.

X-ray examination of the skull on May 22, 1931, showed a depressed comminuted fracture of the left parietal bone. The

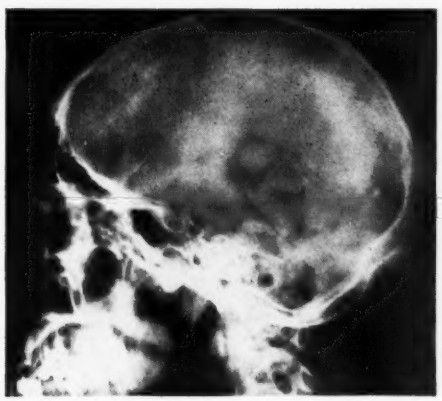

Fig. 19. The fragments have been replaced in a depressed fracture. However, the bone has not been closely approximated. This spacing has not been covered by bone over a period of four years.

fragment was somewhat quadrilateral in shape and wider anteriorly. In the anterior portion it measured 1¾ inches, whereas in the posterior portion it measured 1 inch. The upper fracture extended in almost a straight line from the coronal suture to the lambdoid suture. The lower fracture extended practically the same way with the exception that there was an overriding, especially in its posterior aspect. Another fracture was visible in the inner table about the center of the fragment (Fig. 15). Examination of the skull on April 12, 1933, revealed rounding and fusion of the fragments into one solid mass with even some filling-in of the surface depression (Fig. 16).

As has been previously pointed out by Glaser and Shafer, the early elevation of depressed fractures is not essential in all instances. In particular cases elevation may be entirely eliminated without any danger to the patient whatsoever. The following case represents such an example.

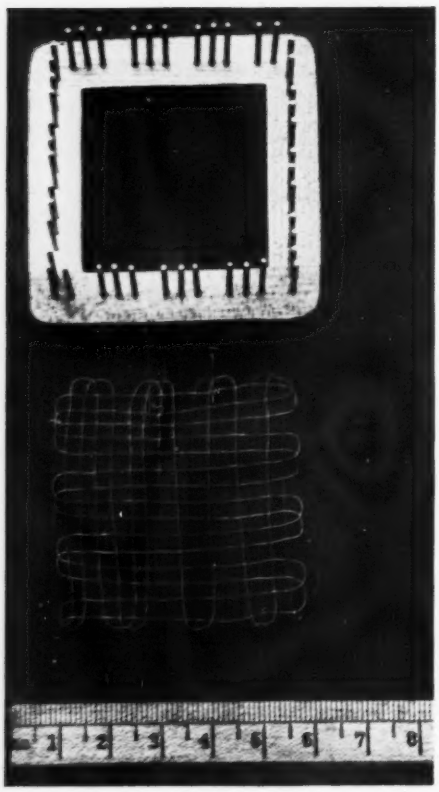

Fig. 20. In order to facilitate surgery a catgut screen has been devised to hold the fragments in place, and also to aid in maintaining position of very small fragments so that complete bony union may occur.

Case 8, G. L., male, aged 25 years, weight 165 pounds, height 5 feet 11 inches, was struck by an automobile, this injury resulting in a dirty, contused, and lacerated scalp, with a deep depression of the skull and a rupture of the dura. Due to the possibility of infection occurring in the wound, it was deemed advisable to remove all fragments of bone. The defect did not decrease in size, and eight months later the serrated edges became smooth.

This patient was injured on May 5, 1936, being rendered unconscious for 30 minutes. He was immediately removed to the hospital. Physical examination was essentially negative, except for abrasions and contusions about the back and shoulder. Neurologic examination revealed a ragged, crisscross, dirty wound in the right temporal region, oozing considerable blood. The examination was entirely negative, save that the patient proved somewhat sluggish in responses. Several x-ray film, taken on May 5, 1936, show a rounded depressed fracture in the right temporal region with the center fragments depressed three-quarters of an inch beneath the surface. These fragments were all removed at operation. X-ray examination one week later revealed a bony defect (Fig. 17), with very slight serration of the edges. X-ray examination eight months later demonstrated a defect of the same size, the only change noticeable being the rounding of the serrated edges (Fig. 18).

It has been the contention of some that the aperture decreases in size, particularly in children. In this series, the youngest patient was six years of age. In this case, x-ray examination eight years later revealed no decrease whatsoever in size. In the case of adults some of these apertures were present as long as thirty years. In carefully checking the size of the apertures, it is well to measure the open-

ing by calipers. In all instances no decrease in size occurred and the only change noticeable was the rounding of the edges, as demonstrated in this case.

Case 9, E. J., male, aged 31 years, weight 175 pounds, height 5 feet 10 inches, was struck on the head by a falling derrick. The primary wound was sutured. Three weeks later the depression was elevated, and the fragments placed *in situ*.

This man was rendered unconscious for a short period of time. He regained consciousness in the ambulance on the way to the hospital. After his entry into the hospital, he again lapsed into unconsciousness and remained so for several hours. Physical examination revealed a rupture of the right tympanic membrane, with bleeding from the right ear. Neurologic examination revealed a lacerated wound in the posterior parietal region, and a depression in the underlying skull. There was some loss of hearing in his right ear. He was in shock. No evidence of intracranial pressure, or focal signs of hemorrhage were shown. He developed a mild otitis media, but the laceration in his scalp healed *per primum*. Three weeks later surgery was performed and the fragments placed *in situ*. The patient returned to work, symptom-free, four months after injury.

Figure 19 demonstrates the position of the fragments one year after injury. There has been no filling-in of the space between the fragments other than the posterior area wherein a certain amount of overlap existed. This region was quite firm to palpation and felt as if the bone was completely united. Films taken in similar cases up to four years after replacement showed a similar spacing.

Realizing the difficulties in placing small pieces of bone in position and maintaining them so that proper healing of the defect may occur, a catgut screen made of woven catgut, as indicated in Figure 20, has been constructed. This screen is sterilized and sewed *in situ*, thus acting as a support for maintaining the position of the fragments and bone dust. By this method, a complete closing of the defect may be obtained. Figure 21 demonstrates close approximation of practically all the fragments in a depressed fracture situated in the frontal region, taken 14 months after injury.

Case 10, W. H., male, aged 51 years, fell 20 feet into a mine shaft. Osteoperiosteal graft was inserted at a later date. Three and a half years after insertion there was only a small defect visible.

This man was dazed, but not unconscious. He had a large laceration in the left occipital region, with a depression of the bone. The case was examined two days after injury and at that time there were no abnormal, neurologic, or physical findings. Due to the fact that the wound was potentially infected and a great amount of débris was situated beneath the scalp, it was deemed good surgical judgment to remove the fragments. He made an uneventful recovery and returned to work three months after injury.

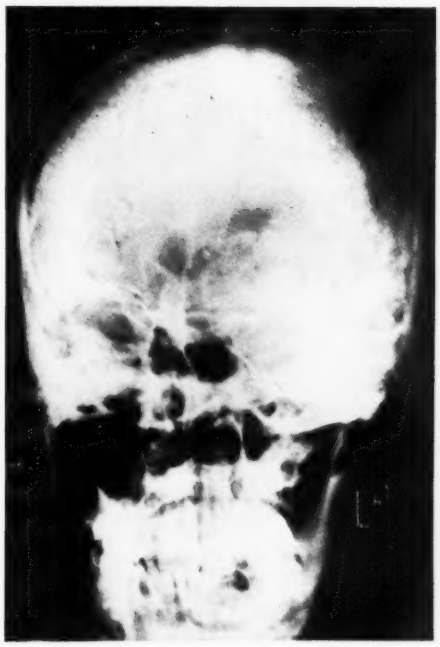

Fig. 21. Closely approximated fragments in the frontal region (right) have united firmly to close the defect almost entirely.

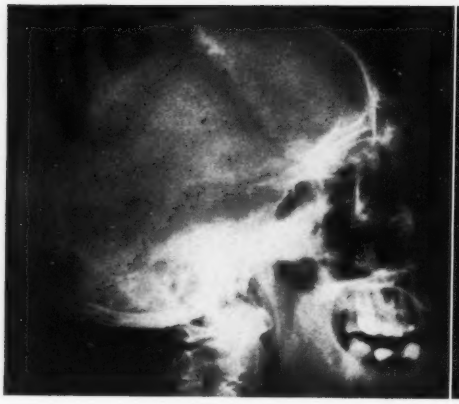

Fig. 22.

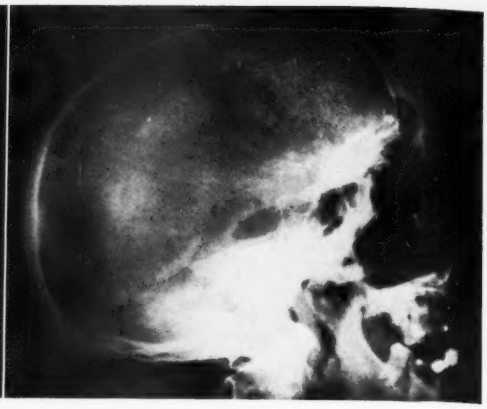

Fig. 23.

Fig. 22. A cranial defect in the parietal region.
Fig. 23. The same patient after an osteoperiosteal graft had been inserted. This film, made three and a half years later, demonstrates the decrease in size of the defect. Larger grafts have taken as long as five years to fill in to this extent.

Later a mild skin infection necessitated treatment of the wound. Due to the mildness of the infection, operative procedure was postponed for a long period of time. On July 2, 1934, the wound was prepared for surgery, but a small pocket of pus was found within the scar. On May 30, 1935, he was again prepared for surgery. X-ray examination at the time of removal of the fragments and in May, 1935, had revealed a cranial defect in the occipital region (Fig. 22). An osteoperiosteal graft was inserted; the patient made an uneventful recovery. X-ray film taken three and a half years later showed a definite decrease in the size of the bony defect (Fig. 23).

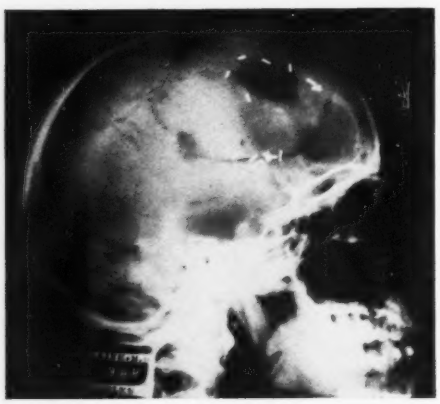

Fig. 24. A bone flap resected around a cranial defect for operative removal of a post-traumatic cyst. This x-ray, taken one and a half years later, reveals a filling in of the cut edges of bone, the normal appearance of the bone within the flap, the defect due to the depressed fracture, the defects caused by burr openings, and the presence of innumerable silver clips placed upon the cerebral vessels when the cyst was removed.

It has been our experience that the use of the osteoplastic graft gives the best results for the closure of the aperture. The introduction of silver plates is extremely dangerous. Infections, or the falling out of the plate, even after long periods, may occur after the procedure. In all of our cases we have been fortunate to have the grafts take, and the apertures gradually become smaller. At all times the graft feels solid to touch and acts as a protective covering to the underlying brain. The good results obtained with these osteoperiosteal grafts taken from the skull, and the simplicity of this procedure, obviates any need to go to other parts of the body for bone.

OSTEOPLASTIC BONE FLAPS

Osteoplastic bone flaps turned down for the removal of brain tumors, hemorrhage,

or other intracranial pathology undergo various types of changes. In infants, before complete ossification of the bones occurs the wounds will heal completely without signs of operative procedure. In adults, the bone flap itself may appear quite normal, or in certain instances calcification within the center of the flap occurs. The edges of the flap and the burr holes may even disappear to a great degree, if close approximation of the flap is carried out and if bone dust is placed in the burr opening. The following two cases demonstrate this.

Case 11, A. S., male, aged 24 years, suffered a depressed fracture from which he developed a post-traumatic epilepsy, secondary to a brain cyst. At a later date a bone flap was turned down to remove the cyst. The bone flap appeared normal two years after injury.

On Dec. 4, 1933, the patient was struck by an automobile and rendered unconscious. He suffered a very severe depressed fracture of the skull, the scalp was lacerated, and the fragments of bone were both deeply embedded in the brain, as well as loosely in the scalp. The dura was badly lacerated and considerable brain tissue oozed from the wound. Due to his precarious condition, emergency measures were instituted, consisting of the removal of the bone fragments and suture of the scalp. He remained unconscious for weeks and hovered between life and death for the first two weeks, yet he eventually recovered. On July 17, 1936, he developed his first convulsion. These convulsions were not of a focal nature. Encephalograms revealed the presence of a brain cyst, and, on June 26, 1937, a bone flap was turned down, and the cyst was resected. Since then he has been entirely free from convulsions. X-ray examination two years later revealed the bone flap in good position, the burr holes quite distinct, and in the posterior edge the healing of the flap to the skull was seen to occur. The central bone defect did not decrease in size, but did not interfere with the circulation of the flap. On the x-ray film (Fig. 24) the numerous silver clips placed in the brain for hemostases may also be noted.

Case 12, E. P., male, aged 28 years, developed a subdural hematoma which was immediately removed. The bone flap, turned down to permit removal of this hemorrhage, degenerated over a period of four years.

This patient had been struck on the head in a hold-up. He was immediately ren-

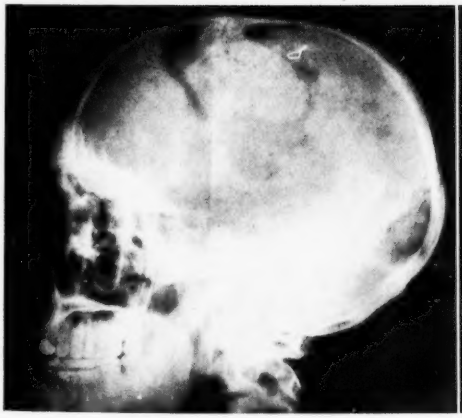

Fig. 25.

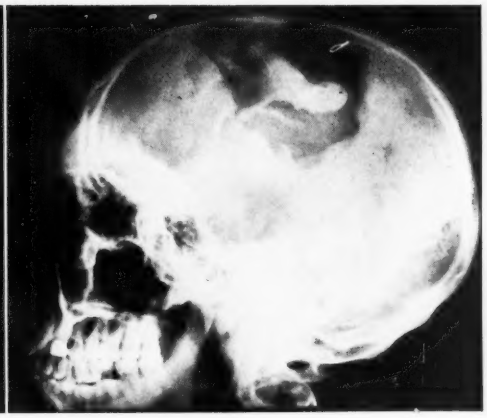

Fig. 26.

Fig. 25. Film taken one and a half years after an osteoplastic flap had been turned down. Note beginning changes about edges of flap and in the center.

Fig. 26. Film taken six years later revealed a marked decalcification in the center of the flap. No further changes had occurred in the six-year period.

dered unconscious and remained so for several hours. Within a three weeks' period, focal signs, as well as evidence of increased intracranial pressure, developed. A diagnosis of a subdural hematoma was made, which was removed by the turning down of an osteoplastic flap. The patient made an uneventful recovery, but one year and a half later developed a generalized epilepsy. X-ray examination at that time (Fig. 25) showed the bone flap *in situ*. In Figure 26, made four years later, there was a generalized decalcification of the bone flap. To palpation, however, this flap was firm to touch. Encephalograms taken upon this patient revealed markedly distorted and irregular ventricles, with an entire shift of the ventricular system. Under barbiturates he has been entirely free from convulsions.

CONCLUSIONS

I. Linear fractures in children under six years of age disappear within six to twelve months after injury, except in instances in which the separation of a fracture has been extremely wide. In those cases the defect may persist permanently.

II. Linear fractures in adults situated in the frontal, parietal, and temporal regions begin to fade immediately. However, complete disappearance rarely occurs under seven months, and in these cases comparison with the original film shows evidence of fracture. The average time of complete disappearance of these fractures, even though fading is evident upon the x-ray film, is three to four years.

III. Fractures in the occipital region take much longer to disappear, and in this series evidence of fracture has persisted as long as eight years, though fading is apparent.

IV. In some instances the area about the fracture absorbs, rather than disappears, leaving a much larger x-ray shadow defect than at the time of injury. This may occur in children as well as adults. The result is a permanent defect.

V. In depressed fractures without elevation, the fragments become rounded and unite, and the lines of fracture cannot be detected, though the depression is apparent.

VI. In operative defects, wherein the bone has been removed, or in cases of depressed fracture wherein the fragments have been removed, the cranial defect never becomes smaller, the only change being a rounding of the edges.

VII. When a bone flap has been turned down around a depressed fracture, and the depressed area carefully hammered out, no evidence of depression or fracture lines will appear upon x-ray examination.

VIII. Bone fragments placed *in situ* at the time of an operation for depressed fracture must be closely approximated, so that all the edges touch, in order to secure complete bony union; otherwise the spaces between the fragments will be visible upon x-ray examination.

IX. Osteoperiosteal grafts are the best means of covering skull defects. They remain firm and solid to the touch and complete calcification depends upon the size of the defect. It requires from five to eight years before the opening of average size is completely calcified.

X. Bone flaps may either undergo absorption, or appear normal.

XI. X-ray films taken long periods after skull injury are just as important in determining the existence of fractures in medico-legal cases as those taken immediately. By a careful study of these films the approximate age of the fracture may be determined in many instances.

DUODENAL ULCER IN THE PRESENCE OF A GALL-BLADDER SYMPTOMATOLOGY

By ERIC J. RYAN, M.D., St. Luke's Hospital, *New York City*

IN its typical form, peptic ulcer gives a symptom complex which runs more true to form than is the case in almost any other condition. The characteristics for peptic ulcer in general may be roughly divided into four as follows: First, meal relation; second, food ease; third, intermittance, and fourth, chronicity.

There is a variation in the symptomatology, particularly as regards food ease, between ulcers in the prepyloric region and those in the postpyloric region. Moynihan (1) has aptly illustrated this in the sequence of pain in relationship to food, in the following manner:

> "In cases of gastric ulcer, the pain which, after an interval, follows the taking of a meal, gradually disappears before the next meal. In cases of duodenal ulcer, the pain continues until the next meal, or until food is taken to give ease to a wearisome pain. The rhythm of gastric ulcer is 'food, comfort, pain, comfort': of duodenal ulcer it is 'food, comfort, pain': a quadruple rhythm in the former disease, a triple rhythm in the latter."

Following the ingestion of food, pain is liable to occur at an earlier time in gastric ulcer than in duodenal ulcer. A characteristic which is frequently noted is the occurrence of night pain, which may come on at the same hour every night. The type of food ingested may cause greater or less pain, the aromatic vegetables and heavy meats producing the most severe type of pain. In duodenal ulcer, particularly, although these same foods may give relief more rapidly, pain tends to return in a more severe form.

In both gastric and duodenal ulceration, a certain number of individuals will give a history which is in no way typical of either condition. In going over a group of cases operated upon at St. Luke's Hospital, New York City, for gall-bladder disease, I was interested in the number in which either mild or moderately severe cholecystitis was noted, with adhesions to the duodenum and the presence of single or multiple ulcers in the adherent area. The interesting question that arises is, which came first, the cholecystitis or the duodenal pathology?

Among many theories as to the cause of peptic ulcer (Nelson, 2), there is mentioned an anatomic relationship between the gall bladder, appendix, and the stomach and duodenum through the related vascular supply of the omentum. The causes of peptic ulcer are generally considered to be thermal, chemical, bacterial, or nervous. Whatever the etiologic factors may be, it is generally accepted that the final stages are (*a*) lowering the resistance of the intestinal mucosa, possibly by altering the production of their protective secretions, and (*b*) allowing digestion of the mucosa by the acid pepsin formed in its own glands.

Ivy and Fauley (3) draw attention to the rôle played by pylorospasm, which, in causing rupture of blood vessels in the stomach and duodenum, may give rise to ulceration. Gastritis and duodenitis may be the forerunners of chronic ulceration.

Among the cases previously mentioned, seven were of particular interest. In them there were certain points which would indicate the possibility that the lesion in the duodenum was secondary to pathology in the gall bladder. In these cases stones were found in the gall bladder, and a rather marked degree of cholecystitis was noted. In four of the cases a moderate degree of cholecystitis was noted with no evidence of stone in either the gall bladder or cystic duct. The absence of stones in the gall bladder with a mild degree of cholecystitis suggests (and the suggestion is borne out by the experience of several surgeons with whom I have had personal communications, 4) that the original pathology in these cases was in

[1] Presented before the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Nov. 28–Dec. 2, 1938.

the duodenum, and by surrounding inflammation and the formation of adhesions had spread to the gall bladder. Case histories in two of these cases and a summary of the findings in the other five illustrate the difficulty in arriving at a correct pre-operative diagnosis in cases of this type.

Case 1. E. T., white female, 37 years of age, whose chief complaint was a pain in the abdomen. Regarding her present illness, the patient states that about 12 years ago, during pregnancy, she had an attack, diagnosed as gallstone colic. She suffered several attacks in the next three months and since then has had several a year. The attacks would come on suddenly and be very severe, the pain radiating to the left side, through the back, and up to the right shoulder. She has had considerable gaseous eructations, but her attacks had no apparent regular relationship to food. The last one left a residual soreness which is still present. She has had no jaundice and no evidence of blood in the stool. Both personal and family histories were negative.

On physical examination the abdomen was tender to palpation in the right upper quadrant; no rigidity nor masses were felt. Other systems were essentially negative. Pre-operative diagnosis was that of chronic cholecystitis and cholelithiasis. On operation the gall bladder was found to be bound with adhesions to the duodenum and showed considerable thickening and contained small stones. The duodenum in its gall-bladder aspect was found to be edematous and the wall indurated near the pylorus. On palpation an ulcer about one centimeter in diameter was found. On opening the duodenum, a kissing ulcer was found on the posterior surface.

Case 2. J. F. white male, 35 years of age, whose chief complaint was epigastric pains after meals for two or three months. About three months previously he began to have pressing, aching pains in the mid-epigastrium, coming on immediately after a heavy meal or about 1 A.M., after a heavy dinner at 7 P.M. The pains occasionally radiated to the left costal margins or up behind the sternum. Sodium bicarbonate (1 teaspoonful in half a glass of water) would be followed by relief from the mild pains in 20 minutes; more severe pains would improve only after vomiting. Attacks occurred more often, he observed, after eating heavy greasy food or soup. Six weeks previously after being wakened every night for a week at 1 A.M. with pain and vomiting, he began eating only light meals. He has been better since, and has had no pain for a week. He was not jaundiced. The stools were not black nor bloody, but light yellow. Personal and family histories were negative.

Physical examination revealed the abdomen to be symmetrical and relaxed. There was slight tenderness in the mid-epigastrium about one inch below the xiphoid, and in the right upper quadrant, beneath the costal margin about one and one-half inches lateral to the midline. There was no spasm, no masses felt. Other systems were negative. An examination of the gall-bladder region showed the gall bladder to fill with the dye. It showed poor emptying in response to food. There were no stones seen. Pre-operative diagnosis was chronic cholecystitis.

Surgical findings revealed the gall bladder to be the site of some adhesions. The wall had much the appearance of chronic cholecystitis; a good many adhesions to the gall-bladder wall both from the duodenum and transverse colon were seen. There were no stones in the gall bladder or common duct. Just beyond the pylorus on the antero-superior surface of the duodenum was a large penetrating ulcer, the surface of which was covered with lymph. The crater of the ulcer measured about three centimeters, and was rather deep.

It will be noted that in the first case the attacks were acute, unexpected, and gave no food relationship. The pain tended to radiate to the back; and whereas radiation of pain in this direction may occur in the presence of peptic ulcer, it is not typical. Typical pain tends to radiate anteriorly, particularly toward the sternum. Of the seven cases mentioned, three showed radia-

tion of pain posteriorly. In the others the pain tended to radiate to the left and upward under the sternum. In none of the cases was there continued food relation, although in two instances it was claimed that pain which followed the evening meal was relieved by the use of sodium bicarbonate.

The duration of symptoms in these patients ranged in order from three months to five years, and from eight to twenty-five years. All of them had periods of relief when no symptoms existed. All showed some of the features of ulceration, but none of them gave the typical complete syndrome. The complaints were such that in only two cases did the clinician feel that an examination of the gastro-intestinal tract was indicated. All had been referred to the X-ray Department for an examination of the gall bladder for pathology. In only one case was the gall bladder visualized. The others showed no filling, and the two cases referred for examination of the gastro-intestinal tract showed duodenal deformity.

An inflammatory process in the abdomen can, and frequently does, give rise to spasm of the sphincters, such as the pylorus, as is seen in the presence of a normal stomach and duodenum with a six-hour gastric retention; which spasm later is shown to be due to disease in either the gall bladder or the appendix. The anatomic relationship, through the circulation and lymphatic distribution between the gall bladder, stomach, and duodenum, is accentuated by the spread of an inflammatory process and the formation of adhesions between them, through which bacterial emboli may be carried in either direction.

An inflammatory process primary in the gall bladder may, by extension to the duodenum and by the spasm set up in the pyloric region, so alter the blood supply and function of the duodenum that an ulcer readily forms. An ulcer primary in the duodenum may likewise, by extension of its inflammatory process to the gall bladder, create a cholecystitis with or without the presence of stones.

It is interesting to note that in the presence of cholecystitis from either cause, the character of the pain, its usual direction, and the general symptomatology of the patient tend to overshadow the typical picture so frequently seen in the presence of gastric or duodenal ulceration.

Conclusions.—An inflammatory process may extend regionally from one organ to another and give rise to a secondary lesion in an adjacent organ; and the clinical picture may be so overshadowed by the symptoms arising from the secondary lesion that the primary lesion will remain clinically unsuspected.

Cases presenting a part, but not all, of the chief characteristics of peptic ulcer, and suggestive of gall-bladder disease, should be examined not only for a gall-bladder lesion, but should be followed by a complete examination of the gastro-intestinal tract.

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DISCUSSION

Nathan B. Newcomer, M.D. (Denver, Colo.): In regard to the frequency of gall-bladder pathology and peptic ulcer in the same individual, I would like to quote from an article by Good and Kirklin, published in the *Collected Papers of the Mayo Clinic*, of 1936. In an analysis of 100 cases of peptic ulcers, they found 80 normally functioning gall bladders. In addition, there were eight normally functioning gall bladders in which they were unable to rule out stones; two normally functioning gall bladders with tumors; three normally functioning gall bladders with stones; two poorly functioning gall bladders; one poorly functioning gall bladder with stones; two non-func-

tioning gall bladders, and two non-functioning gall bladders with stones. In seven of the 100 cases of peptic ulcer, the gall bladders were reported to be functioning abnormally.

Skinner, Lockwood, and DeWeese state:

"The presence of chronic deforming pathology upon either side of the pylorus without peritoneal irritation does not influence the roentgen gall-bladder test, but the presence of active pathology with peritoneal irritation or active penetrating lesions may interfere with the usual response of a normal gall bladder to the tetraiodophenolphthalein oral capsule test."

In a paper on the mobility of the gall bladder,[1] we showed that the normal gall bladder moved upward and outward from 1.5 to 2 in. after taking a full meal of solid food, and that failure to do so was practically invariably accompanied by gall-bladder pathology.

In several previous papers we have discussed the relation of the antrum and the cap to the gall bladder and have shown that any alteration in the intimate relation of the antrum and cap to the gall bladder interferes with the normal functioning of the gall bladder.

Consequently, I believe that a duodenal ulcer with peritonitis or adhesions which limit the mobility of the gall bladder will sooner or later end in stagnation in the gall bladder and in cholecystitis. I do not believe the gall-bladder pathology is due to direct spreading of the infection from the antrum to the gall bladder.

On the other hand, adhesions in the upper right quadrant due to gall-bladder pathology are common. No reasonable explanation occurs to me as to why this should cause a duodenal ulcer. However, until we have a final accepted explanation of the cause of duodenal ulcer, this phase of the question cannot be decided.

We all know that about 20 per cent of duodenal ulcers are symptomless: also, that cholecystitis, even with stones, may give few or no symptoms.

The pain of duodenal ulcer may be due to hyperacidity, to hyperperistalsis, to obstruction, or to accompanying peritonitis. Soda relief in an uncomplicated duodenal ulcer should be immediate; that is, not over one minute. Soda relief in from 15 to 20 minutes indicates some other pathology and the relief is due to emptying of the stomach. The history of such a case should be carefully taken, particularly concerning the beginning of the attacks and any variation in this history should be carefully noted.

Gall-bladder attacks appear at irregular periods, lasting a few days. I doubt that an attack which occurs every day for several weeks is due to gall-bladder pathology.

In the first case reported by Dr. Ryan there is a clear-cut history of gall-bladder disease dating back to pregnancy twelve years before. Dr. Ryan stated that the patient's last attack had left a residual soreness which was still present. This point in her history should have made one suspect a change in the original pathology.

In the second case, the chief complaint was epigastric pain after meals for two or three months. Such daily pains are generally not due to gall-bladder pathology and call for a gastro-intestinal examination. Dr. Ryan is absolutely right when he states that if the case is not characteristic of peptic ulcer and is suggestive of gall-bladder disease, there should be a complete gastro-intestinal examination. It is my personal belief that we do entirely too many incomplete abdominal examinations. A proper gastro-intestinal examination should include a gall-bladder examination and *vice versa*.

B. A. RHINEHART, M.D. (Little Rock, Ark.): It is not necessary to tell roentgenologists that the complete examination of all the hollow abdominal viscera must be made in order to establish a diagnosis. Thirty-eight years ago, Dr. McCarrison, of the British Indian Army Service, em-

[1] NEWCOMER, NATHAN B., and NEWCOMER, ELIZABETH H.: The Mobility of the Antrum, Pylorus, Duodenum, and Gall Bladder in Health and Disease: The Influence of Mobility in the Functioning of These Organs in the Biliary Tract. RADIOLOGY, **28**, 339-350, March, 1937.

phasized the fact that none of the degenerative infections and functional disorders of the gastro-intestinal tract was found in the hill tribes of northern India. Checking on that statement, I have found that in other parts of the world, in selected primitive peoples, there also is an absence of all these conditions, notably in Persia, Liberia, the Belgian Congo, the Hawaiian Islands, and elsewhere. On the other hand, Buddhists of India who eat no meat and drink no milk have a high frequency of gastro-intestinal disorders.

In these disorders, you will find two or more conditions which have previously been called "entities," occurring in a great number of patients. These entities may be appendicitis, cholecystitis, peptic ulcer, or purely functional disorders known as a spastic colon, mucous colitis, gastritis, etc.

As far as pain from hollow abdominal viscera is concerned, I think it has been fairly well established by Pottinger, in California, by Bolton, of England, and by Best and Taylor, of Toronto, that such pain is all due to increased muscular tension. In working out this question, years ago I started making routine tests of the neural muscular irritability in all gastro-intestinal patients, and, with the exception of 2 per cent who seemed to react abnormally, practically every one of these patients had an abnormal neuro-muscular irritability as determined by the galvanic current with Erb's test.

Kaufman has said that he believes spasm is the cause of duodenal ulcer. Perhaps there are other factors concerned with that, but spasm, I believe, is the reason ulcer does not heal so readily.

The symptomatology of gall-bladder disease is quite confusing. So far as I am concerned, there is only one symptom of gall-bladder disease and that is jaundice. In his work, Evarts Graham found that 40 per cent of patients do not recover from their symptoms following cholecystectomy; 30 per cent will obtain partial relief. The remaining 30 per cent are relieved. Relief can be assured only in cases in which there is colic due to stone. However, there may be colic in the upper right abdominal quadrant due to a spasm of Cannon's sphincter and distention of the hepatic flexure with gas. Therefore, roentgenology is the only method of diagnosis in these disorders of the gall bladder, and I believe Dr. Graham would agree with me on that.

MAURICE FELDMAN, M.D. (Baltimore, Md.): The subject of association of peptic ulcer and gall-bladder disease is interesting. In a recent study of 115 cases of peptic ulcer in whom the symptoms were not characteristic of peptic ulcer and who had had pains in the right upper quadrant with gas, distention, etc.—symptoms referable to the gall bladder—cholecystographic studies revealed that 87.8 per cent of the gall bladders were normal and that 10.4 per cent revealed a non-filling or poor filling gall bladder.

The percentage of non-filling seemed to be rather high in a normal gall bladder, and we felt that perhaps there was some reason for this non-filling, such as associated gall-bladder disease or the effects of hyperacidity upon gall-bladder visualization.

Many years ago Dr. Friedenwald, Dr. Kearney, and I studied a large series of cases for the effect of hyperacidity on the filling of the gall bladder. We did not find a single instance in which the gall bladder failed to fill but we did frequently find a shadow of lessened density.

In the series of 115 cases, 10.4 per cent showed a non-filling gall bladder. In 27.3 per cent of these we observed a poor or sluggish contraction. Dyskinesia of the gall bladder is a common condition and in cases of ulceration we have either a retarded or accelerated evacuation of the gall bladder.

Although we attribute these changes to dyskinesia of the gall bladder, as Dr. Ryan has pointed out, many of these cases may be due to associated pericholecystitis or other gall-bladder conditions.

Formerly we have believed that we should obtain a normal gall-bladder shadow in every case of hyperacidity, but, in recent

years, in giving the larger doses of dye, or the double dose of dye, if you choose to, our cases of lessened density in peptic ulcers have been markedly diminished and we now obtain better densities of gall-bladder shadows. However, I wish to emphasize again that in cases in which we do not obtain a good gall-bladder shadow, a large percentage of these show an associated gall-bladder pathology.

It is interesting, too, that even by the intravenous method, as Lieb has pointed out, 26 per cent of organically normal gall bladders failed to fill in 100 cases of duodenal ulcer. This figure seemed to be rather high.

I am inclined to believe that dyskinesia of the gall bladder is due to its close proximity to the duodenum, causing associated symptoms as a result of cholecystic duodenal adhesions or excessive irritability.

ERIC J. RYAN, M.D. (*closing*): The subject under discussion interests me because it is, as I previously said, an incomplete examination, and for accurate preoperative diagnoses, we have to convince the clinicians of the necessity of a complete examination.

We frequently encounter these cases. In my abstract I have changed, a little, the intent of my paper; I did so purposely to give rise to discussion. I think when we have indefinite findings in cases of this type we should insist more and more on as complete an examination as possible.

MILLIAMMETER INACCURACY CAUSED BY CONTROL CABINET[1]

By MARVIN M. D. WILLIAMS, PH.D., *Rochester, Minnesota*

From the Division of Physics and Biophysical Research, the Mayo Foundation

WHILE calibrating an x-ray therapy machine it was noticed that the milliammeter in the aerial system read about 10 per cent higher than the milliammeter on the control cabinet which was in the grounded circuit of the secondary of the high voltage transformer. The aerial milliammeter was checked with a laboratory meter, battery, and rheostat, and found to furnish correct readings to within ±1 per cent. The milliammeter in the control cabinet was checked with the same laboratory meter, both with the battery and while the x-ray machine was in operation, and was found to furnish readings that were 10 per cent too low. When the meter was removed from the control cabinet the readings differed from those of the laboratory instrument by only 1 or 2 per cent. Bringing a piece of iron or steel up to the meter caused the reading to decrease, the amount of the decrease depending on the size, shape, and position of the metal.

The control cabinet meter has a bakelite case and the top of the control cabinet in which the meter is mounted is of steel. The steel surrounding the meter acts as a shunt for the meter's magnet, thus reducing the strength of the magnetic field around the coil of the meter and causing the reading to be low. The effect seems to be constant, since the calibration of the meter has not changed in the course of a year. Some meters in metal cases were found to be affected by iron in the same way, although the effect was much smaller than for meters in bakelite cases.

If the intensity of the radiation is measured with a suitable dosimeter, a constant error in the milliammeter reading may be of little importance, although it will give a false value of the output per milliampere. A more serious result of such an error is the danger of overloading the x-ray tube if the tube is being used at or near its full capacity. The operation of a tube at a few per cent above its rated capacity may not do any serious harm, but it is entirely possible that some meters might be affected by the presence of iron or steel so that the readings would be more than 10 per cent low, and such an overload on the tube would undoubtedly reduce its life. In view of this fact, it would seem to be advisable to check the meters without removing them from the position in which they are to be used, and the cause of any difference in the reading of the meter in the aerial system and the meter on the control cabinet should be carefully investigated. On some machines, depending on the type of rectification, the checking of the control cabinet meters would have to be done with alternating current instead of with a battery.

[1] Submitted for publication in June, 1939.

CONGENITAL CYSTIC MALFORMATION OF THE LUNGS, FORMERLY KNOWN AS "VESICULAR" OR "HYPERTROPHIC PULMONARY EMPHYSEMA," RECENTLY DESCRIBED AS "CONGENITAL CYSTIC DISEASE OF THE LUNGS," WITH REPORT OF A CASE[1]

By CHARLES TILLEY SHARPE, M.D., *New York City*

RECENT articles published on congenital cystic disease of the lungs have applied a new name to a well known and long established clinical and pathologic entity. True, x-rays have permitted the visualization of the cysts in the living subject, but they have added nothing to the excellent clinical description of the disease given by Laënnec (26) under the heading "Symptoms and Signs of Vesicular Emphysema" and also by Sir Thomas Watson (48) and others.

A study of emphysema reveals that large cysts were reported by Matthew Baillie (2), Theophile Bonet (6), Giovanni Battista Morgagni (34), Van Swiéten (46), Sir John Floyer (16), Ruisch (Friederich Ruysch), 40, and Antonio Maria Valsalva (45).

Jean Baptiste Bouillaud (7) mentioned a case in which the bladder or pouch was equal to the size of a stomach of ordinary dimensions.

More recently Charles Hilton Fagge (14), James Copland (11), Rindfleisch (37), Rokitansky (39), and Virchow (47), have reported interesting observations. For example, Fagge (14) writes:

"There is an enormous destruction of capillaries when the affection is at all extensive. Rindfleisch speaks of the vessels as collapsing until 'only a narrow ribbon-like band is left, which may be recognized as an obliterated vessel by its greater transparency amid a dark, often pigmented, parenchyma, and by its uniting with other bands like itself to form the usual anastomotic network.' "

He goes on to say that—

"Some relatively wide communications are opened up between the pulmonary artery on the one hand, and the pulmonary and bronchial veins on the other hand. These anastomoses appear in well injected lungs as peculiar elongated, unbranched, vascular arches of the same diameter throughout, contrasting very strikingly with a far more numerous assemblage of extremely tortuous and dilated arteries, for whose contents no such supplemental mode of escape has been provided."

Is this not an earlier description, and a satisfactory explanation, of the "fine curvilinear shadows characteristic of a multilocular cyst," described by Kirklin (24) and by Schenck (41), and visible in the case reported herewith?

As to autopsy findings, the description given by Laënnec (26) of one of his own cases is as fine a description as one is likely to find on this subject. Watson (48) gives a generalized description which agrees in many features with that of Koontz (25), as reported by Kirklin (24), and also with the description given by Schenck (41). The congenital origin of the anomaly did not escape the observation of these men, and there are arguments for and against it by some of the authors mentioned in the bibliography. For example, Sir William Osler (36) wrote:

"On inquiry we find that these patients have been short-breathed from infancy, and they belong, I believe, to a category in which there has been a primary defect of structure in the lung tissue."

Virchow (47) was inclined to believe in the existence of a primary disease of the pulmonary tissue which developed in early life and which became more marked as time went on. He described the loss of pigment and termed it "albinism of the lungs."

The freedom of these cases of pulmonary vesicular emphysema from tuberculosis was noted by Louis (28).

The involvement of the right side of the heart in congenital cystic disease of

[1] Accepted for publication in April, 1939.

the lungs was referred to recently by Willius (49). It was described by Laënnec (26) and more extensively by Watson (48).

Many theories as to this condition were advanced and experimental work to determine the cause dates back to 1879, when Lichtheim (27) recorded his observations in the *Archives of Experimental Pathology*. More recently, Cervello (9), Hirtz (21), Friedman and Jackson (17), Harris and Chillingworth (19), and, still more recently, Hinshaw (20) report the production of subpleural emphysematous vesicles with a ball-valve apparatus, thus confirming the theory advanced by Matthew Baillie (2).

From Fantanus (15), who described "a large bladder of air which communicated with a bronchi in the lungs of a markedly cyanotic infant of three months," quoted by Meyer (33), to the case here reported, the findings are so much alike as to size, position, number, structure, contents, color within and without, time of origin, method of formation, and as to their communicating with a bronchus and at times occurring as isolated cysts, that there can be no doubt the same condition was being observed. Reports of the clinical findings by some authors, notably Laënnec (26), are so remarkably accurate that they leave no question that cases of congenital malforma-

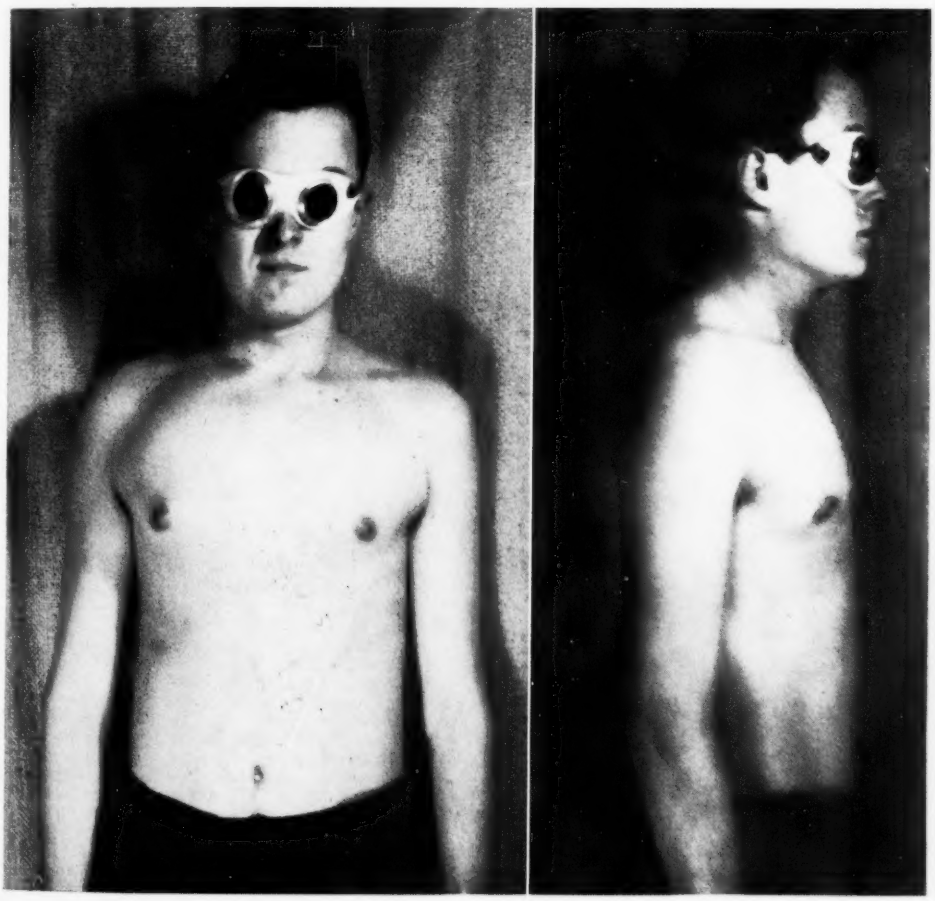

Fig. 1. The patient. Note the distention of the upper chest.
Fig. 2. Lateral view of the patient. Note the position of the spine in the upper dorsal area.

tion of the lungs were observed and well understood long before the advent of the x-ray.

Congenital cystic disease of the lungs is more descriptive than *pulmonary emphysema*, but not as satisfactory as *congenital cystic malformation of the lungs*. In selecting a new name for an old and well known condition we should not forget the dictum of Samuel Bard (4), one of the organizers of the College of Physicians and Surgeons of New York City, namely, "New names are mostly deceiving, new theories are mostly false or useless, and new remedies for a time are dangerous."

The adoption of a new name is not only often deceiving, but worse, in that it threatens the loss of the most interesting

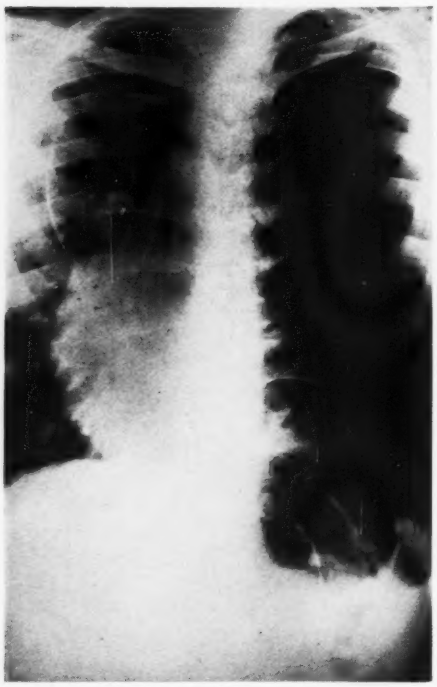

Fig. 3. Teleoroentgenograph, postero-anterior position, during inspiration. The original film shows a marked translucency of the entire left side with curvilinear lines running in different directions, most marked longitudinally with a concavity toward the mediastinum. Quadrilateral shaped air cells are visible at the base. The entrapped air in some of these cells may be seen fluoroscopically to be behind and below the stomach. The cells change their shape with respiration. The diaphragm on the left side is not well defined because of the grouping of these air cells and the air in the fundus of the stomach. The trachea is clearly outlined to the right of the sternum. The bronchus is visible. Its branches descend almost perpendicularly to the right of the spine. The left border of the heart is hidden behind the sternum. A well defined band is seen to extend from the upper right area of the heart shadow upward toward the clavicle, terminating at the level of the second rib anteriorly on this side. This is the right border of a cyst in the right side of the chest. The axis of the ribs on the left side is more horizontal than on the opposite side.

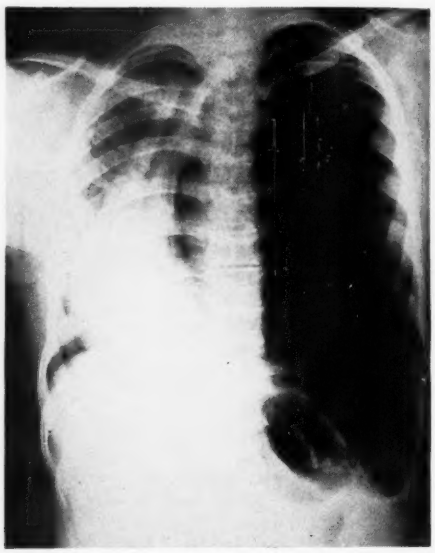

Fig. 4. A teleoroentgenograph of the chest in the postero-anterior position, during full expiration. Note the displacement of all the contents of the right chest further to the right. The heart is much closer to the side of the cage—evidence of the pressure of the entrapped air in the left side. The curvilinear lines have changed their former position and the air cells at the base of the left chest are less quadrilateral and are extended in length. The band running from the upper right heart shadow is not visible in this film. The bifurcation of the trachea is visible and the difference in length of the right and left branches is quite evident. There seems to be some rotation of the contents in the right chest. The entrapped air is visible to the right of the spinal column. The left border of the pericardial shadow is concave. The right border of the pericardium is convex and is much closer to the side of the cage than in inspiration. During expiration the diaphragm is relatively much higher on the right than on the left, while during inspiration the two sides are about equal. The shadow of the bronchus and the aorta are confused and practically indistinguishable. This film shows a concavity of the diaphragm in the left side near the border of the cage. It is 12 cm. below the diaphragm on the right side. The compression of the lung tissue is very marked during expiration.

and valuable literature that has been compiled by our predecessors. Unless it is recognized that *congenital cystic disease of the lungs* has been known for centuries as *vesicular pulmonary emphysema*, we stand to lose as valuable a literature as we do by substituting *encephalitis* for *edema of the brain*. We might better follow the example of the dermatologists who hold fast to everything, *capillis capitis exceptis*. They add as many names as they wish in the description of dermatologic lesions, but they retain the old ones, thus preserving a complete bibliography.

Finally, if by the adoption of this new name for an old condition we sacrifice the masterful descriptions of vesicular emphysema, we may deceive our successors and limit their perspective as much as we would by failing to recognize that the great advance in our knowledge, that of visualizing the cystic changes in the lungs in life,

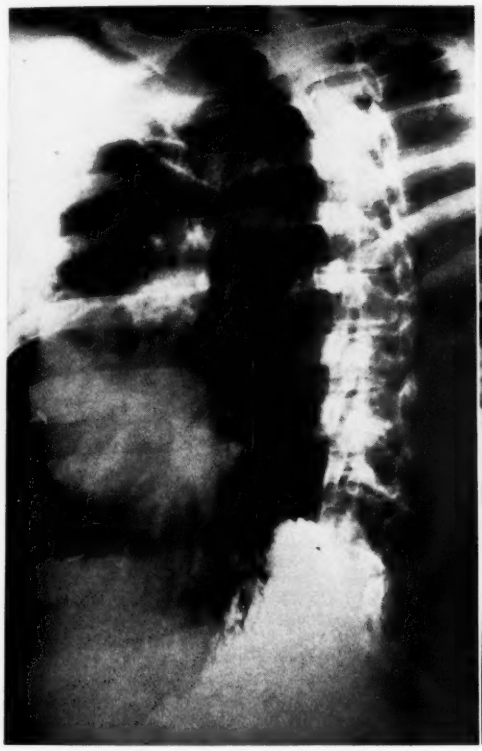

Fig. 5.

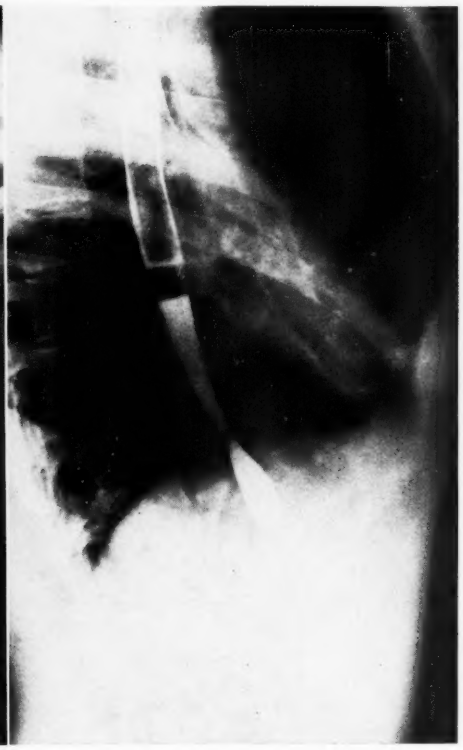

Fig. 6.

Fig. 5. This film was taken with the Potter-Bucky diaphragm and with the patient recumbent and in a semi-oblique position. It shows displacement of the heart shadow to the right, with a concavity in the upper area of the heart shadow due to an air cyst above it. The trachea is well defined. The bifurcation is clearly seen. On the right the bronchi are visible for a considerable length and appear to descend almost perpendicularly in the chest. The branch which appears to go to the left side is very short and seems to terminate within the entrapped air space before reaching quite to the right side of the spinal shadow. Other shadows visible in the original film suggest that the lower branches of the bronchus do not pass further to the left than the main one noted above. There is no evidence of ordinary lung markings in the left side of the chest. The axis of the ribs and their spacing are indicative of entrapped air under considerable pressure. Barium is visible in the stomach.

Fig. 6. A lateral view with the right side to the cassette. The esophagus is outlined with barium which is seen passing into the stomach. The diaphram is well defined and intact. The ballooning of the chest is evident. The upper part of the sternum bulges outward and upward, and the inner surface is markedly concave. The ribs are almost horizontal. They are widely spaced, as evidenced by their relation to the vertebral column on the left side as shown in the film. A large air cyst is visible anterior to the great vessels and the bronchi.

is due not to the work of Roentgen alone, but to a long line of scientists who devoted their attention especially to electrical research.

The bibliography which follows does not pretend to be exhaustive. It supplements in part that of Kirklin and that of Schenck.

REPORT OF A CASE

P. D., white male, examined Dec. 22, 1936; age 20 years; height 5 ft. $11\frac{1}{4}$ in.; weight $138\frac{1}{2}$ pounds. Chest measurements: inspiration, 38 in.; expiration, 36 in. Abdominal measurement, 29 in. Pulse rate, 100. Blood pressure: systolic 110, diastolic 80.

Clinical Pathology.—Blood studies: hemoglobin, 94 per cent; red blood cells, 5,152,000; white blood cells, 7,600; polymorphonuclears, 75 per cent; lymphocytes, 17 per cent; eosinophiles, 3 per cent; large monocytes, 5 per cent; sedimentation rate, 5 mm. in one hour.

Urinalysis: normal.

History.—Measles at age two; otherwise negative for infectious diseases. He had some trouble with the sacro-iliac joints at age twelve or thirteen and wore a brace for one year. The pain in the back was so severe that he would fall in the street because of it.

The first indication of the developmental defect was when he started to school and it was noticed that he could not run as fast as other boys of his age. After extreme physical effort he would fall, and at times vomiting would occur. When he recovered his breath, he was all right. He has never had a serious illness.

Examination of Chest.—The outstanding symptoms and findings are barrel-shaped chest, dyspnea and cyanosis on exertion. Tactile fremitus and breath sounds are absent on the left side. Vocal fremitus is heard faintly, evidently transmitted from the right side. Râles are present at times at the right base. Excursion of diaphragm is about one inch. On fluoroscopic examination, movement of the diaphragm is seen to be accompanied by a kaleidoscopic shifting of the entrapped air present in the stomach and the air cells at the left base, some of which are below and behind the fundus of the stomach. The heart is displaced to the right side. It shifts markedly with respiration.

When examined the patient was a "runner" for a financial institution and attended evening classes several times a week. When taking part in theatrical performances he found it hard to speak his lines because of the necessity of breathing frequently. With all his handicaps, he enjoys good health and a remarkable freedom from colds and upper respiratory infection.

Every clinical detail exhibited by this case can be duplicated in the writings of the authors quoted above. The only new feature is the visualization of these cysts

Fig. 7. A view of the left side of the chest taken from the same film as shown in Figure 3 in an effort to bring out the curvilinear lines more clearly.

in the living subject, thanks to the development of the x-ray.

120 Broadway

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CAVITARY BRONCHOGENIC CARCINOMA[1]

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CHARACTERISTIC features of primary carcinoma of the bronchus are sufficiently well known to-day so that with the use of modern methods the diagnosis is in general established readily. In many instances, however, the findings so closely simulate chronic pulmonary infection that the possibility of cancer may be overlooked. This is especially true of those bronchogenic carcinomas which cavitate, since both clinically and roentgenologically they may resemble pulmonary abscess and tuberculosis. Inasmuch as carcinomas of the lung may now be amenable to surgical treatment, their exact differentiation is of practical importance. It was thus considered worthwhile to review the pertinent clinical and roentgenologic aspects of this particular type of lesion. In the American literature, special attention has been given the cavitating bronchogenic carcinomas in the reports of Fishberg and Rubin (3) and Reisner (7).

The frequency with which carcinoma of the bronchus cavitates has been pointed out by numerous pathologists from a study of autopsy material. For example, Rosedale and McKay (6) found abscess cavities in 18 of 37 autopsied cases; Jaffé (4), in 12 of 100 cases, and Fishberg and Rubin (3) in 15 of 51 cases. Koletsky (5), in a study of 100 autopsied cases, described cavitation 4 cm. or greater in diameter in 30 instances.

The pathogenesis of cavitation in primary bronchogenic carcinoma can be understood from a consideration of certain of its pathologic features. Necrosis is characteristic of nearly all tumors, resulting either from growth of the tumor disproportionate to the accompanying blood supply, or to vascular changes in or about the neoplasm. It is frequent in bronchogenic carcinoma to find the larger pulmonary vessels in the region of the hilum either compressed or invaded by tumor. Both in and around the carcinoma, smaller vessels may be found occluded by thrombi or emboli, often composed of tumor cells. These changes may, therefore, produce aseptic necrosis of tumor tissue by decreasing the blood supply. Since carcinoma of the lung arises so frequently in association with the larger bronchi, this necrotic material can be eliminated readily from the lung, leaving the pulmonary excavation.

In addition to tumor necrosis, infection also plays a significant rôle. It is well known that areas of lung distal to points of bronchial constriction are subject to various inflammatory changes. The retention of secretion facilitates invasion of the lung by pyogenic organisms from the upper respiratory tract and mouth, with the production of chronic pneumonitis and suppuration. Carcinoma of the bronchus, a frequent cause of bronchial obstruction, is prone to develop these complicating changes with suppurative necrosis of the involved tissue. As in abscess or tuberculosis, this material is expectorated with consequent cavity formation.

In occasional instances, infection of the tumor itself may be negligible, the predominant changes occurring entirely in the adjacent lung parenchyma. In these cases, the production of bronchiectasis or abscess may completely obscure the underlying carcinoma which remains undiagnosed.

The exact rôle played by necrosis of tumor tissue or infection may be difficult to determine; both mechanisms are probably involved in the majority of instances. Nevertheless, certain features may aid in

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differentiation. Tumor necrosis with cavitation is most apt to occur in those carcinomas which are infiltrative in character with a massive lobar distribution. Grossly, these cavities appear to have a thick irregular wall, the lining of which is shaggy, friable, and apparently composed of tumor tissue. The appearance suggests that the central portion of the tumor has sloughed, leaving behind a circumferential rim of carcinomatous tissue. This finding is borne out by the microscopic examination which reveals tumor at every point in the cavity wall without significant inflammatory or suppurative changes.

Cavities produced largely through infection tend to be more localized, their walls thinner and more regular, with the appearance frequently of a pyogenic membrane. The cavity contents are more purulent or fluid in character, and, depending upon the nature and extent of the infection, may be either putrid or non-putrid. Gangrene is not, in our experience, a common occurrence. Microscopic examination of the cavity wall may reveal only suppurative changes, and, it may be necessary to examine numerous sections of the wall to discover the carcinomatous infiltration.

Cavitation occurs in all types of bronchogenic carcinoma but most frequently in the squamous-cell variety. In Koletsky's (5) series, 52 per cent of squamous-cell carcinomas, 27 per cent of adenocarcinomas, and but 9 per cent of small-cell carcinomas revealed cavity formation. Similar findings are cited by Atkin (1) and Reisner (7).

Clinical Aspects.—This study is based upon a review of 127 cases of primary bronchogenic carcinoma admitted to Cleveland City Hospital from 1927 to 1937, inclusive, proved by autopsy, bronchoscopic or operative biopsy, and in which roentgenologic examination was performed. In 15 individuals (12 per cent), the roentgen demonstration of cavitation necessitated the differentiation from tuberculosis and lung abscess.

In this group the patients were all males and the ages varied from 42 to 68 years, with ten patients in the fifth and sixth decades. This finding is consistent with the age and sex distribution of all bronchogenic carcinomas at this hospital, since they predominate in males by a ratio of 9:1, while 60 per cent occur between the ages of 40 and 60 years.

A review of the histories of these 15 individuals failed to reveal any clinical picture which could be considered pathognomonic of a cavitary bronchogenic carcinoma. The important symptoms were those common also to tuberculosis and lung abscess. Cough, expectoration, loss of weight, and weakness occurred in all patients. Irregular fever above 37.5° C. was noted, at some time during the course of the illness, in 13 patients. Hemoptysis occurred in 12 cases and pain was present in 10. Five patients had clubbing of the fingers.

Although the symptomatology is usually insufficient to differentiate conclusively between these cavitating pulmonary lesions, certain aspects of the clinical picture should at least suggest the possible presence of carcinoma, particularly in individuals above the age of 40 years.

In this group of patients, the onset of the illness was insidious, with an irritative cough as the initial symptom. In eight cases, the cough was non-productive for periods as long as from three to eight months and only later was purulent expectoration noted. This latent period before the onset of expectoration is of considerable value in the differentiation from lung abscess, since patients with the latter lesion will rarely remain sputum-free for this length of time. We have not encountered a clinical picture simulating that of an acute lung abscess.

There was no type of expectoration which would be considered characteristic of a cavitary bronchogenic carcinoma. Unlike most lung abscesses, however, the daily sputum volume was small and seldom exceeded 30 c.c. of purulent material. In contrast to pulmonary abscess, foul expectoration was noted in but two patients.

Pleuritic pain is common to all cavitating lesions of the lung. In these patients with

cavitary carcinomas, however, the pain frequently appeared early, and was constant, severe, and non-pleuritic in type. Such persistent, localized pain which fails to respond to medication is unusual in abscess, and particularly in pulmonary tuberculosis, and should suggest the existence of a carcinoma.

In this group of patients, nothing of differential diagnostic value could be observed from the presence of fever, hemoptysis, weakness, fatigue, and weight loss. The physical signs elicited were variable and usually insufficient to establish the diagnosis. Obvious signs of pulmonary atelectasis and cavitation were significantly lacking. The most important finding consisted of decreased breath sounds and voice sounds in the involved area, indicating the presence of some degree of bronchial obstruction. However, similar signs accompanied by râles and impairment of the percussion note may also occur in abscess and tuberculosis. Nevertheless, in the presence of roentgenologic evidence of cavitation, diminished or absent breath sounds should indicate the possibility of an underlying carcinoma.

As in all forms of bronchogenic carcinoma, other findings may suggest the correct diagnosis. Particularly important will be the demonstration of Horner's syndrome, obstruction to mediastinal blood vessels, vocal cord paralysis, lymph node and subcutaneous metastases. However, since cavitary bronchogenic carcinomas are most frequently of the squamous-cell variety, extra-thoracic metastases will not be commonly found (5).

At the time of admission to the hospital, the clinical picture presented by this group of patients was that of a chronic, steadily progressive illness in its terminal stages. Eleven of the 15 individuals died within two months of admission. In ten of the 15 patients, the total duration of life from onset of symptoms was less than one year. One patient lived 20 months after the initial symptoms.

Roentgenologic Features.—Roentgen signs in cavitary bronchogenic carcinoma include all features observed in other types of cancer of the bronchus, with the addition of a cavity. The roentgenogram usually shows a unilateral mottled area of increased density, roughly circular or ovoid in configuration, and containing a rarefied area in or near its central portion. The extent of the density constituting the cavity wall varies from a thin line (Fig. 5) to many centimeters in thickness (Fig. 1). The ratio of the size of the cavity to the thickness of the wall depends upon the degree of necrosis and sloughing of the tumor. In our series, the signs of atelectasis were more often absent than present. Homogeneous dense areas due to pleural effusion may be an accompanying feature. The tumor cavity often contains a horizontal fluid line, usually in its base. In those instances in which the cavity cannot be recognized on the film, fluid fills the cavity completely and gives the appearance of a consolidated area similar to inflammatory or neoplastic infiltration. More frequently, however, fluid is expectorated through a communicating bronchus and the cavity remains only partially filled. A film made with greater penetration, with the Potter-Bucky diaphragm or with planigraphic technic, is often required to demonstrate the details of the cavity. The location of the tumor is variable; in our group, the cavity was situated in the right lower lobe in six instances, the right upper lobe in three, the left upper lobe in two, the left lower lobe in two, the right middle lobe in one, and along the left interlobar septum in one. If extensive, the cavity may involve more than one lobe of the lung, especially if it originates in the base of either upper lobe. Lesions near the median plane of the chest which appear on the single postero-anterior film to be located near the hilum are, in most instances, situated in the apex of a lower lobe (Cases 3 and 8). Because of the difficulty of determining the exact location of the lesion in the postero-anterior view, lateral or oblique projection is often required. Occasionally, the cavity is situated distal to the neoplasm when it does not represent the tumor (Fig. 14). The size

of the cavity ranges from the smallest diameter visible on the films to that of an entire lobe. The average size in 15 patients was 5.2 cm.; the smallest being 1.5 cm. and the largest 14 cm. in diameter. In lesions situated peripherally, with extension to surrounding structures, gross rib destruction may be observed (7). As in any type of bronchogenic carcinoma, enlarged lymph nodes may be present in the hilum as a result of regional metastasis. The cavity simulates that found in pulmonary abscess and gangrene, tuberculosis, or bronchiectasis. In the presence of a cavitary lesion of the lung, roentgenologic evidence of metastasis in bones, in brain or other soft-tissue parts may be of aid in establishing the diagnosis. Bronchograms, by demonstrating filling defects or occlusion of the lumen of the bronchus leading to the cavity, will give the clue to the diagnosis of neoplasm.

DIFFERENTIAL ROENTGENOLOGIC DIAGNOSIS

Lung Abscess.—It is almost impossible to differentiate the cavitary form of primary bronchogenic carcinoma from the ordinary type of pulmonary abscess on roentgenographic findings alone, since the appearance of cavitation with surrounding consolidation in either instance may be exactly alike. In the serial examination of lung abscess, the cavity may become gradually smaller and the consolidation resolve, while in carcinoma the lesion becomes progressively more extensive. Temporary regressions of the tumor may take place in exceptional instances (Figs. 6, 7, and 8).

Pulmonary Tuberculosis.—Carcinoma is usually a unilateral disease, whereas tuberculosis more often involves both lungs when it reaches the stage of cavitation. Mottling is usually more extensive in tuberculosis, especially in the lung which contains the cavity. Multiple cavitation is more frequent in tuberculous than in carcinomatous lesions.

Empyema.—Chronic encapsulated pockets of purulent fluid with communicating bronchial fistulæ may simulate cavitating lung tumors. Such pockets are usually located in the base of the thoracic cavity or in the periphery of the lung-fields. The shadow is usually fusiform or semi-oval in outline and is situated next to the pleural lining. Mottling is absent around the cavity. Signs of bronchial stenosis are absent.

Echinococcus Cyst.—If single, an echinococcus cyst may be confused with neoplasm (2). The cyst is smooth-walled, without infiltration in the surrounding parenchyma and without evidence of bronchial stenosis. Other foci of hydatid disease are usually present in the liver and other portions of the abdomen.

Diagnostic Procedures.—From the preceding discussion, it is apparent that in cavitary bronchogenic carcinomas neither the clinical nor the roentgenologic features are sufficiently distinctive to establish an exact diagnosis. The extent to which a correct and early diagnosis will be made must depend upon the alertness of the clinician in the utilization of the available diagnostic procedures. Early diagnosis is particularly important in this type of bronchogenic carcinoma since it is so frequently of the squamous-cell variety. Koletsky's (5) studies indicate that the squamous-cell carcinoma is slow-growing, locally invasive, and infrequently metastasizing, hence offers the best possibility for successful surgical intervention.

In our 15 patients, a correct diagnosis of carcinoma of the bronchus was made in ten instances. In one case all diagnostic procedures failed to reveal the true nature of the disease and the diagnosis was based upon the clinical course. Diagnosis was established by bronchoscopic examination in six patients and by biopsy of the lung at thoracotomy in three others. In one of the latter cases, however, the patient was considered to have a lung abscess for several months until biopsy from a deep portion of the cavity wall revealed carcinoma. Of the five incorrectly diagnosed cases, one was called sarcoma of the lung, three lung abscesses, and one pulmonary tuberculosis. In the last individual, it was known from

biopsy of a lymph node that carcinoma was present but the lung was not seriously considered as the primary source.

Bronchoscopic examination is undoubtedly the most valuable confirmatory procedure. In four of the cases erroneously diagnosed, study of the autopsy specimens indicated that bronchoscopy would probably have revealed the correct diagnosis. It has thus become an invariable rule in this hospital that all cavitary lesions of the lung which cannot be definitely established as tuberculous in origin should be examined bronchoscopically to aid in excluding the presence of an underlying carcinoma. This is especially important in males above the age of 40 years, with the clinical picture of a chronic lung abscess. Bronchoscopy will be of least assistance in diagnosing carcinomas situated peripherally or in the upper pulmonary lobes. Thus in two patients with carcinomas of the upper lobe, the trachea and bronchi appeared normal.

Occasionally, microscopic study of portions of the cavity wall removed at thoracotomy for drainage of an abscess may be of great value. In three patients the diagnosis was established in this manner. It must be remembered, however, that in those carcinomatous cavities resulting particularly from infection, the wall may show only suppurative changes and the specific section removed for examination may not reveal the presence of carcinomatous infiltration. In those instances in which abscess formation occurs entirely peripheral to the carcinoma, the cavity wall may not contain tumor tissue and the diagnosis thus remains obscure. Case 8 illustrates an example of this occurrence.

Biopsy of lymph nodes and subcutaneous nodules and the examination of pleural fluid for tumor cells may occasionally provide the clue to the correct diagnosis. Aspiration biopsy of the pulmonary lesion under fluoroscopic guidance should also be

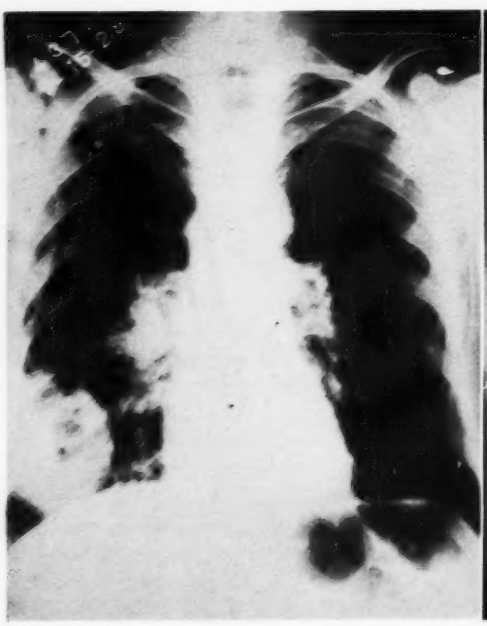

Fig. 1.

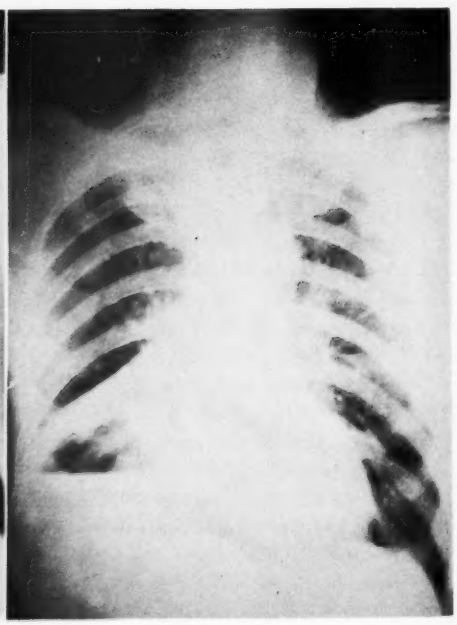

Fig. 2.

Fig. 1. Case 1. Large round mass in right base with central rarefaction representing cavity. Enlarged lymph nodes in right hilum. Original diagnosis: metastatic sarcoma. Autopsy: poorly differentiated adenocarcinoma with metastases to right hilum and right upper lobe.

Fig. 2. Case 2. Large cavity in right base with fluid level which has the appearance of a large pulmonary abscess in the lower lobe.

considered. We have employed this procedure frequently, although, in this particular group of patients, no positive results were obtained.

Repeated examination of the sputum for tubercle bacilli should always be carried out, since it will be of assistance in excluding the commonest cavernous lesion of the lung. It must be remembered, however, that carcinoma and tuberculosis may occasionally co-exist. Examination of the sputum for tumor cells was employed twice in this series as a diagnostic procedure although with negative results.

Bronchography has proven of limited value. In those instances in which evidence of bronchial constriction was present there was uncertainty whether this resulted from inflammatory change or carcinoma. The demonstration of bronchial constriction by instillation of iodized oil should, nevertheless, suggest the possibility of bronchogenic carcinoma.

ILLUSTRATIVE CASES

Case 1. J. G., a 52-year-old white male, was admitted on Aug. 3, 1928. In December, 1927, he had developed a severe cough. Two weeks later he had expectorated bloody sputum, and this recurred until the time of admission. He had lost considerable weight and complained of marked weakness. Chest pain was absent.

The patient appeared emaciated. The significant findings consisted of dullness in the right upper chest and scattered râles in the infrascapular region. The sputum was negative for tubercle bacilli.

A roentgenogram of the chest (Fig. 1) showed a circular shadow of increased density measuring 7 cm. in diameter in the lower portion of the right lung, probably in the middle lobe. In the upper portion of the shadow there was an oval rarefied area which had the appearance of a cavity. The right hilum was enlarged to 4 cm. in diameter. A small faint nodule 1 cm. in diameter was situated in the upper right lung opposite the level of the second intercostal space anteriorly. The impression was that of metastatic sarcoma.

The patient's condition was terminal at the time of admission and death occurred on Aug. 29, 1928, nine months after the onset of the symptoms. The temperature varied between 37° and 39° C.

The clinical diagnosis was that of lung tumor, probably metastatic in character. No other primary tumor could be discovered.

Autopsy (Dr. Herbert Reichle) revealed a primary carcinoma of the right middle lobe with metastases to the right upper lobe, diaphragm, right hilum lymph nodes, right adrenal, right kidney and bones. Microscopically, the tumor was a poorly differentiated adenocarcinoma. The right middle lobe consisted of a well encapsulated mass of white tissue, 10 cm. in diameter, containing a completely necrotic center.

Comment.—The smooth circular mass in the right base with the accompanying small nodule in the right upper lobe led to the erroneous diagnosis of metastatic sarcoma. Cavitation should have suggested the presence of a primary tumor, since it is rare, in our experience, to find cavitation in pulmonary metastases.

Case 2. T. P., a 49-year-old white male, was admitted on Jan. 14, 1932, complaining of a non-productive cough of several years' duration. Three months before admission, this became more marked and was accompanied by purulent expectoration. In addition, persistent pain had been present for two weeks in the right chest posteriorly. The temperature ranged between 37 and 39° C.

The percussion note was impaired over the right lower chest. Breath sounds were decreased in intensity, but numerous coarse râles were present.

Fluoroscopic and film studies of the chest (Figs. 2 and 3) showed a large cavity, 10 cm. in diameter, occupying most of the right lower lobe, but situated more posteriorly than anteriorly. The cavity was half-filled with fluid which was seen to shift under the screen while changing the patient's position. The remainder of the right lower lobe was consolidated.

The patient was considered to have a lung abscess of the right lower lobe, which was surgically drained on Jan. 22, 1932.

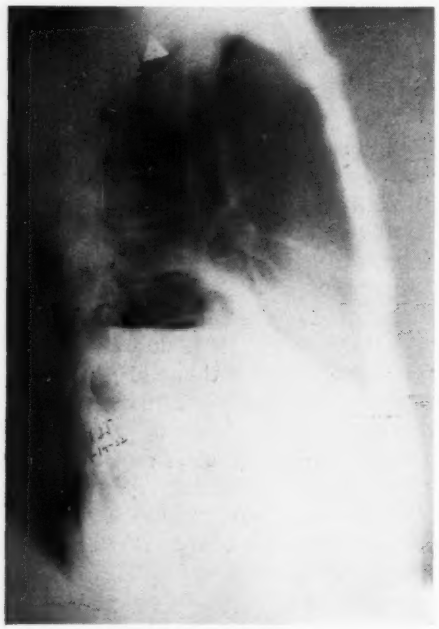

Fig. 3. Case 2. Lateral view of the chest showing a large cavity with fluid level in the posterior portion of the right lower lobe. Biopsy of the wall of the cavity showed a well differentiated squamous-cell carcinoma.

Culture from the abscess material showed gram-positive cocci and *Staphylococcus albus*. Biopsy of the cavity wall showed no evidence of carcinoma.

One month later the patient showed marked weakness, dyspnea, and loss of weight. He continued to have purulent expectoration. Partial thoracoplasty was carried out on June 2, 1932. On Oct. 10, 1932, because of his failure to improve, a biopsy was secured from the deeper portions of the abscess and this tissue revealed the presence of a well-differentiated squamous-cell carcinoma. The patient continued to lose weight, with fever ranging between 38 and 39° C. daily. Death occurred on Nov. 12, 1932, eleven months after admission to the hospital.

Autopsy (Dr. Theodore Wille) revealed a cavity 7 cm. in diameter in the right lower lobe (Fig. 4). The wall of the cavity was composed of grayish-yellow friable tissue.

Microscopically, the tumor was a well-differentiated squamous-cell carcinoma of the right main bronchus, with metastases to the regional nodes and pericardium.

Comment.—This case is representative of the type in which an original diagnosis of lung abscess was made because of the presence of a large cavity and purulent sputum. Biopsy of the cavity wall at the first operation did not reveal tumor tissue. The correct diagnosis was proven only when biopsy from the deeper portions of the abscess showed the presence of squamous-cell carcinoma. Cancer should have been suspected clinically, because of the insidious onset with a non-productive cough, in a middle-aged male, the diminished breath sounds, persistent chest pain, and failure of the patient to improve after drainage of the abscess. From the location of the tumor at autopsy, it is probable that bronchoscopic examination would have revealed the presence of carcinoma.

Case 3. J. B., a 67-year-old white male, was admitted on Sept. 7, 1933. In November, 1932, he developed cough and expectoration of small amounts of bloody purulent sputum. These symptoms were followed by weakness, persistent pain in the right chest, and weight loss of 33 pounds. Because of the clinical symptoms and the roentgen appearance of cavitation, the patient was thought to have pulmonary tuberculosis. Artificial pneumothorax had been started at another hospital.

The patient had an emphysematous chest with râles in the lower half of the right lung. The sputum was negative for tubercle bacilli. There was no elevation in temperature.

Fluoroscopic and film examination of the chest, on Sept. 12, 1933 (Fig. 5), showed a small pneumothorax on the right. There was a circular shadow of diminished density in the apex of the right lower lobe, measuring 4 cm. in diameter, due to a cavity. The infiltration forming the wall

of this cavity was thin, measuring 2 to 3 mm. in thickness.

Ten days after admission the patient died following a profuse pulmonary hemorrhage.

The clinical diagnosis was lung abscess.

The autopsy diagnosis (Dr. T. T. Frost) was squamous-cell carcinoma of the right lower lobe bronchus, with cavitation and metastases to the right bronchopulmonary and tracheobronchial lymph nodes. The lower lobe showed a 5 cm. cavity filled with blood clot and having a granular wall. Microscopically, the wall was composed of tumor tissue which invaded the surrounding bronchi, vessels, and lung parenchyma.

Case 4. H. B., a 53-year-old white male, was admitted on Nov. 28, 1934. In January, 1934, the patient developed a non-productive cough, with weakness and fatigue. In July, 1934, following the occurrence of several small hemoptyses, with a loss of 15 pounds in weight, the patient was admitted to Sunny Acres Sanatorium. During the previous month he had expectorated small amounts of purulent sputum which was not foul. Because of these symptoms and the presence of a cavity shown by roentgenographic findings, he was believed to have pulmonary tuberculosis. The sputum was persistently negative for tubercle bacilli. Since he failed to improve, he was transferred to City Hospital for further investigation.

The patient appeared emaciated. There were a few râles over the upper portion of the left lung. On the back of the neck there was a small non-tender nodule fixed to the skin. Biopsy of the nodule revealed an undifferentiated carcinoma.

Postero-anterior roentgenogram of the chest, on July 23, 1934 (Fig. 6), showed a thin-walled cavity about 4 cm. in diameter in the lateral portion of the middle third of the left lung-field. Both hili were unusually prominent. Chest films, on Sept. 8, 1934 (Fig. 7), and Nov. 7, 1934 (Fig. 8), showed a marked diminution in the size of the cavity which measured 1 × 3 cm. and was surrounded by a ring-like infiltration of greater thickness. The last examination, on Dec. 12, 1934, revealed a dense mottling, 4 cm. in vertical dimension, extending from the left hilum outward to the lateral chest wall, and a scattered nodulation from the level of the second rib to the diaphragm. There was no cavitation at this time.

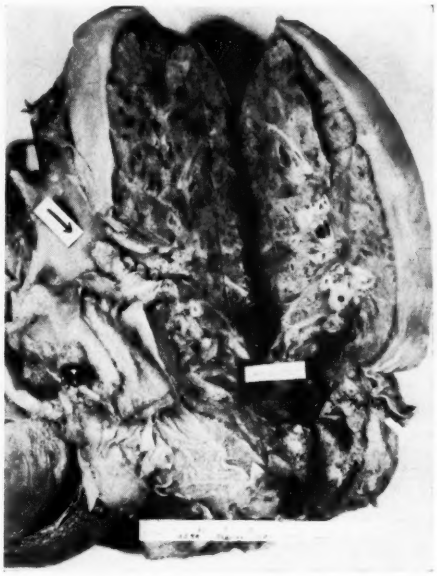

Fig. 4. Case 2. Pathologic specimen showing a huge abscess in the right lower lobe and a tumor in the right eparterial bronchus.

The patient's temperature was normal. He continued to expectorate small amounts of purulent sputum. Examination revealed acid-fast bacilli on two occasions. He failed rapidly and death occurred on Jan. 29, 1935. The final clinical impression was that of pulmonary tuberculosis and carcinoma of undetermined origin. The lung was considered a possible but not proved source of tumor.

Autopsy (Dr. Mae Gallavan) revealed a small-cell carcinoma of the left lung, with metastases to the mediastinal, mesenteric, aortic, axillary and cervical lymph nodes, pancreas, spleen, and kidney. The left lung showed a firm area, 5 × 6 cm. in dimension, along the interlobar septum, containing a 2.5 cm. cavity filled with

yellow mucoid material. Tumor tissue extended along the interlobar septum to the hilum where a white mass appeared which spread into the lung in a radial manner along the bronchi and blood vessels. About 2 cm. from the carina there was slight constriction of the left main bronchus. There was no evidence of pulmonary tuberculosis.

Comment.—Cases 3 and 4 were originally diagnosed as pulmonary tuberculosis because the roentgenogram showed the presence of thin-walled cavities. Case 4 was more confusing because of the reported tubercle bacilli in the sputum and the diminution in the size of the cavity. Nevertheless, the possibility of carcinoma was suggested by the patient's progressive course, disproportionate to the roentgenologic extent of his disease, and since it was known from the biopsy of the skin nodule that he had an undifferentiated carcinoma. In Case 4 it is doubtful that bronchoscopic examination would have been of aid in the diagnosis.

Case 5. M. O., a 50-year-old white male, was admitted on June 8, 1935. Three months previously the patient had developed a non-productive cough which was attributed to a "chest cold." After several weeks he began to expectorate mucopurulent sputum which was never foul. On two occasions small hemoptyses occurred. There was persistent pain in the left upper chest. He had lost ten pounds in weight and experienced fatigue readily. Shortly before admission his sputum had become scanty.

There was slight impairment of the percussion note over the left upper chest. The breath sounds were of good quality, although slightly decreased in intensity. There were coarse râles anteriorly and posteriorly. The sputum was negative for tubercle bacilli.

A postero-anterior roentgenogram of the

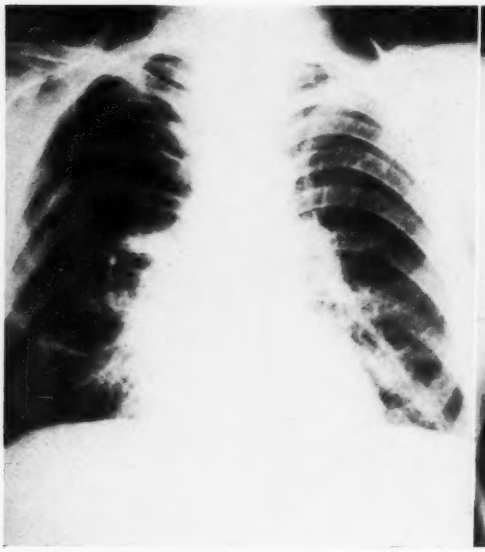

Fig. 5.

Fig. 6.

Fig. 5. Case 3. A large thin-walled cavity, 4 cm. in diameter, at the level of the right hilum, was diagnosed as lung abscess. The actual location was in the apex of right lower lobe posteriorly Autopsy diagnosis: squamous-cell carcinoma.

Fig. 6. Case 4. Roentgenogram made on July 23, 1934. Arrows point to thin-walled cavity, 3 cm. in diameter, in the left upper lobe, which was diagnosed ulcerative pulmonary tuberculosis in the early course of the disease. Prominent hili.

chest on July 6, 1935 (Fig. 9), showed a dense mottling in the upper left lung-field occupying an area 7 × 10 cm. The margins of this shadow were irregular. There was a rarefied area centrally located and

Comment.—This case showed a thick-walled cavity in the upper lobe of the left lung, appearing roentgenographically as an abscess. The diagnosis of carcinoma was suspected clinically but not proven until

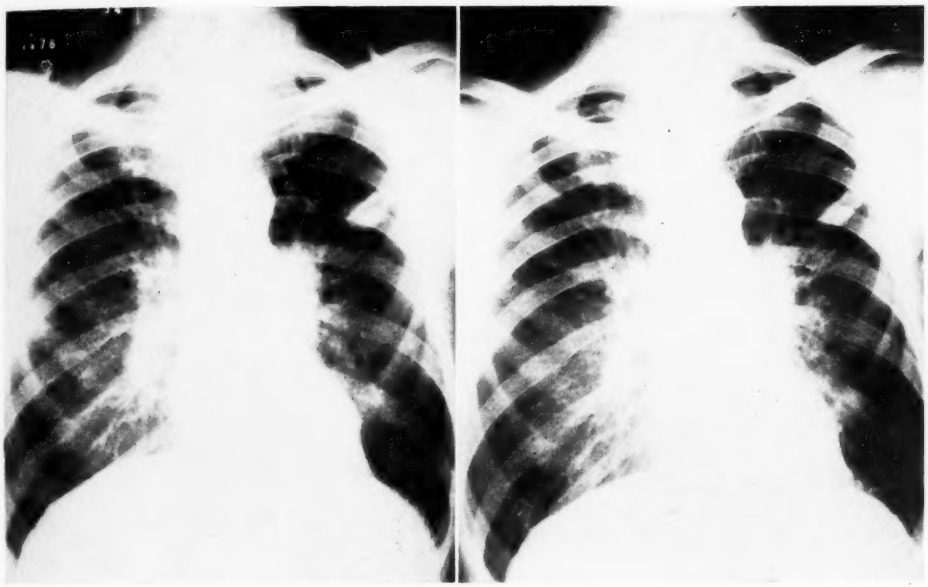

Fig. 7. Fig. 8.

Fig. 7. Case 4. Roentgenogram made on Sept. 8, 1934, in which the cavity in left upper lobe is shown to be smaller than in Fig. 6, and surrounded by a thick ring of infiltration. The left hilum is unusually prominent.

Fig. 8. Case 4. Roentgenogram made on Nov. 7, 1934, shows the cavity to be considerably smaller than that observed on previous examination. There is a slight decrease in the thickness of the wall. Autopsy: small-cell carcinoma of the left lung with metastasis to mediastinum.

with a definite fluid level indicating the presence of a cavity.

The patient's temperature varied between 37 and 38.4° C. Because of the roentgen findings the patient was considered to have a lung abscess, possibly on the basis of a bronchogenic carcinoma. Bronchoscopy showed no evidence of intrabronchial tumor. Drainage of the abscess was performed by a two-stage operation on July 23 and July 30, 1935. Biopsy of the wall of the abscess showed the presence of a small-cell carcinoma. Following the operation, the patient failed to improve and died on Aug. 3, 1935, six months after the onset of symptoms. No autopsy was performed.

biopsy of the cavity wall revealed a small-cell carcinoma. The presence of the initial non-productive cough and chest pain were significant clinical findings.

Case 6. H. S., a 65-year-old white male, was admitted to the tuberculosis pavilion on Sept. 18, 1935, complaining of shortness of breath, cough, expectoration, loss of weight and strength for the previous three and one-half years.

The patient appeared slightly emaciated. The chest was emphysematous and the breath sounds were distant, but not abnormal. On auscultation there was prolongation of the expiratory phase. Post-tussive râles were heard at the left apex posteriorly. The temperature varied be-

tween 37 and 38° C. The sputum was negative for tubercle bacilli.

Previous roentgenographic examination of the chest on March 1, 1935, showed a streaky soft mottling in the lateral portion of the left subapical region at the level of the second intercostal space anteriorly. The appearance was that of minimal pulmonary tuberculosis. There was a moderate increase in the extent of infiltration on June 6, 1935. Because of the enlargement of the left hilum shadow, increase in the extent of the infiltration to an area 5 cm. in diameter (Sept. 24, 1935), and the appearance of large hilum lymph nodes in the lateral roentgenogram, the diagnosis of primary carcinoma of the bronchus with regional lymph node metastases was considered. The right anterior oblique view on Oct. 8, 1935 (Fig. 10), and the postero-anterior view on Nov. 9, 1935 (Fig. 11), showed a definite cavity with a fluid level in the center of the consolidation. On Jan. 23, 1936, ten days before death, the consolidation occupied the upper third of the left lung-field.

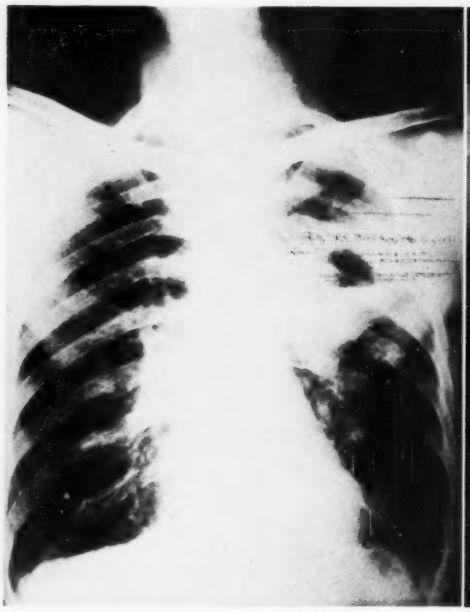

Fig. 9.

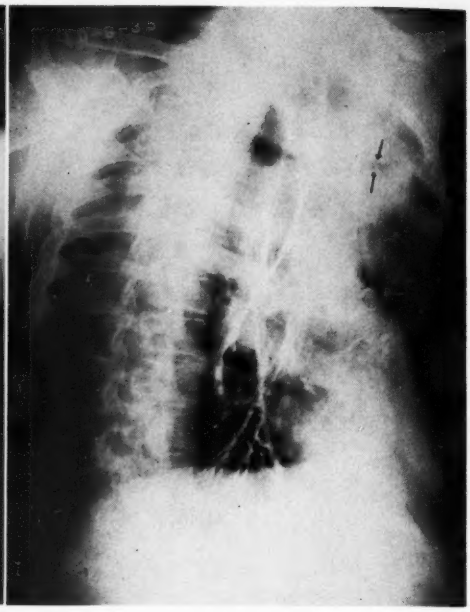

Fig. 10.

Fig. 9. Case 5. A cavity, 5 cm. in diameter, containing fluid and surrounded by dense infiltration in the right upper lobe. Clinical and roentgen diagnoses: lung abscess. Biopsy of the wall of the abscess showed small-cell carcinoma.

Fig. 10. Case 6. Right anterior oblique view of the chest (Oct. 8, 1935) showing a small cavity, 1.5 × 3 cm., in the left subapical region surrounded by dense infiltration. At this time the cavity was not visualized in the postero-anterior view.

Bronchoscopic examination on Oct. 8, 1935, was negative. Iodized oil failed to enter the left upper lobe. Aspiration biopsy was attempted on two occasions without success. The patient developed bradycardia, suggesting metastasis to the brain. He became extremely nervous and confused mentally. Death occurred on Feb. 2, 1936. The final clinical diagnosis was carcinoma of the bronchus.

Autopsy (Dr. A. Chernoff) revealed a poorly differentiated adenocarcinoma of the left eparterial bronchus with cavitation and metastases to the left bronchopulmonary lymph nodes, right adrenal gland, and brain. In the left upper lobe a cavity

6.5 cm. in diameter was found (Fig. 12). There was a soft mass growing down into the bronchus from the edge of the cavity about 4 cm. from the left main bronchus.

Comment.—Case 6 represents a thick-walled cavernous lesion of the lung. The patient was admitted with a diagnosis of pulmonary tuberculosis because of the original roentgenographic appearance. However, the enlargement of the left hilum, the persistent negative sputum, and the progressive course established the diagnosis of bronchogenic carcinoma. Bronchoscopy, bronchography, and aspiration biopsy all failed to indicate the true nature of the lesion.

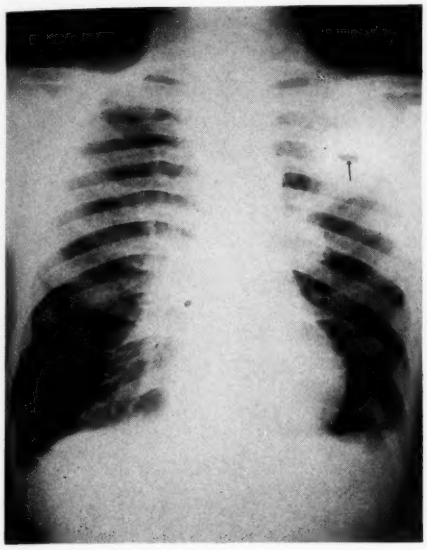

Fig. 11. Case 6. (Nov. 9, 1935.) A large circular area of increased density, measuring 4.5 cm. in diameter, in the left subapical region showing at this time a small cavity in its central portion. The arrow points to fluid level. (See postmortem specimen.)

Fig. 12. Case 6. The postmortem specimen showing large round tumor in the left apex. The central portion of the neoplasm contains a cavity the walls of which are lined with necrotic material. Microscopic examination revealed a poorly differentiated adenocarcinoma of the eparterial bronchus.

Case 7. A. C., a 54-year-old white male, was admitted on July 17, 1936, complaining of severe cough for the previous five and one-half months. There was no purulent expectoration or hemoptysis. For five weeks previous to admission, the patient had had pleuritic pain on the right side. The patient showed evidence of weight loss and had a persistent irritative dry cough. The chest was dull to percussion over the lower half of the right lung posteriorly. Breath sounds were absent and scattered râles were heard in the right midlung anteriorly and posteriorly. The temperature was 39° C. The sputum was negative for tubercle bacilli. On aspiration, 775 c.c. of thin purulent fluid with a specific gravity of 1.022 were obtained. No organisms were seen on the smear. Histologic examination of the pleural fluid revealed no tumor cells.

Thoracotomy on July 28, 1936, was followed by considerable improvement. The patient was lost to observation until Aug. 3, 1937, when it was found that copious foul expectoration had developed,

with 20 pounds loss of weight and constant pain in the right chest. Thoracentesis produced 60 c.c. of pus. Surgical drainage of a large bronchiectatic cavity in the right lower lobe was done on Aug. 7, 1937.

pleural effusion. Bronchograms made on Jan. 21, 1937 (Fig. 13), showed cylindrical dilatation of the lower lobe bronchi as a result of bronchiectasis. No significance was attributed to the constriction at the origin

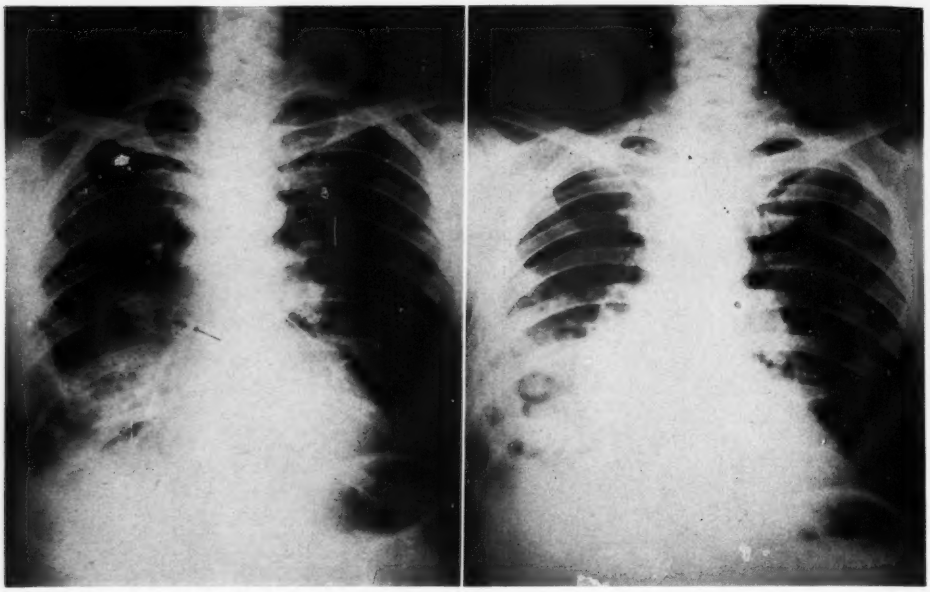

Fig. 13. Fig. 14.

Fig. 13. Case 7. (Jan. 21, 1936.) Instillation of iodized oil in the right lower bronchial tree. Arrows show constriction of the right descending bronchus at the site of the carcinoma. Lower bronchi show cylindrical bronchiectasis. Inflammatory reaction in the right lower lobe in the area of the surgically drained abscess.

Fig. 14. Case 7. (Aug. 4, 1937.) A large cavity with fluid level in the right lower lobe, posteriorly, is shown. The cavity represents an abscess distal to the neoplasm which is situated at the proximal portion of the right main descending bronchus. Resection of the right rib in posterior axillary line.

Biopsy of the pleura and underlying lung showed chronic inflammation. The patient's condition became progressively worse and he expired on Oct. 12, 1937.

Roentgen examination of the chest on July 16, 1936, revealed a dense homogeneous cloudiness throughout the lower half of the right lung-field with an ill defined upper border, indicating the presence of fluid and consolidation. After partial resection of the ninth right rib, a small pneumothorax was present with a moderate collapse of the right lower lobe. On Nov. 17, 1936, the cloudiness in the right base extended higher laterally than medially and represented a re-accumulation of the

of the right descending bronchus. Postero-anterior and lateral views on Aug. 4, 1937 (Figs. 14 and 15), showed a large cavity in the right base with an extensive amount of infiltration surrounding it.

Autopsy (Dr. Simon Koletsky) showed the fistula in the right chest to lead into a large irregular cavity occupying the postero-lateral portion of the lower lobe, a portion of the middle lobe, and a small portion of the upper lobe adjacent to the hilum. The fistula extended into the pericardium. A tumor mass involved the right lower lobe bronchus about 1 cm. from its origin and extended into the pulmonary tissue to the eparterial bronchi. Micro-

scopic examination showed a poorly differentiated squamous-cell carcinoma of the lower lobe bronchus with extension into the right lung, the superior and inferior bifurcation lymph nodes, and pericardium.

bronchus on the bronchogram was masked by the cylindrical bronchiectasis. Bronchoscopy should have been performed and would have revealed the nature of the lesion.

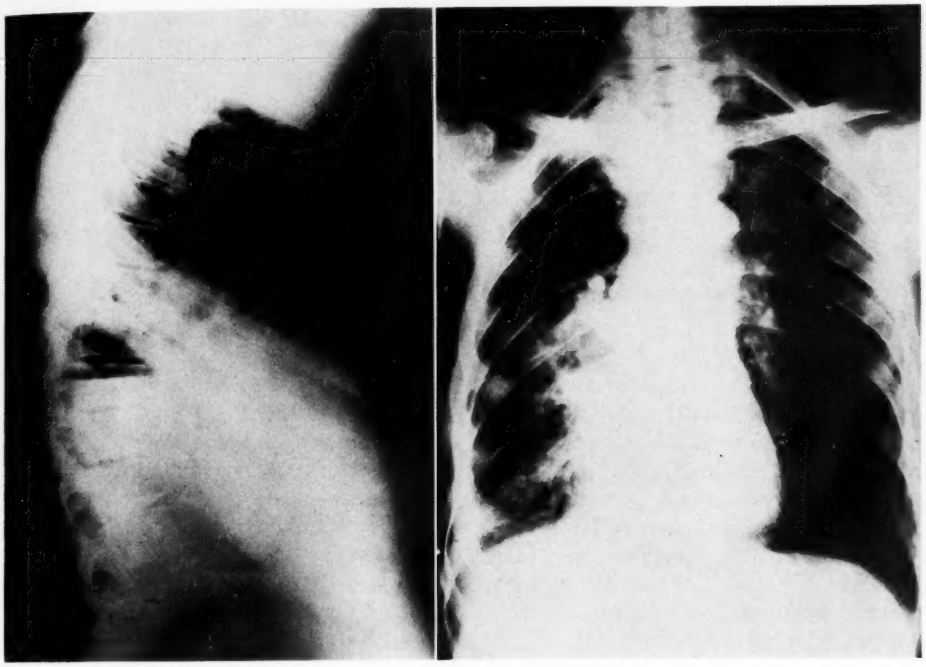

Fig. 15. Fig. 16.

Fig. 15. Case 7. (Aug. 4, 1937.) Lateral view showing a large cavity with fluid level in the posterior portion of the right lower lobe. Abscess distal to the carcinoma.
Fig. 16. Case 8. A cavity, 4 cm. in diameter, with fluid level, situated in the apex of the right lower lobe. (See gross specimen in Fig. 17.)

The cavity wall revealed tumor infiltration only in its medial portion. The lateral wall adjacent to the site of operation showed no tumor tissue.

Comment.—Case 7 illustrates the type of case in which the abscess cavity lies entirely peripheral to the carcinoma and hence obscures it. In these instances it can be understood why biopsy of the cavity wall may not be of diagnostic value. Carcinoma was not suspected in this individual although the insidious onset with non-productive cough, decreased breath sounds, and the progressive course suggested its presence. The significance of the constriction of the right lower lobe

Case 8. J. S., a 68-year-old white male, was admitted on July 25, 1936. In February, 1936, the patient had developed a cough with slight blood-streaked sputum. Since that time he had lost 40 pounds in weight and complained of weakness.

Examination showed the patient to be emaciated. There was slight retraction of the right supraclavicular fossa. The breath sounds in both lungs were obscured by sonorous râles.

A postero-anterior view of the chest on July 27, 1936, showed a cavity 2 cm. in diameter in the right hilum region and dense mottling in the medial portion of the lower third of the right lung. On Sept.

Fig. 17. Case 8. Autopsy specimen showing large cavity in the apex of the right lower lobe. Microscopic examination showed a well-differentiated squamous-cell carcinoma.

15, 1936 (Fig. 16), re-examination of the chest including a lateral view showed the cavity to be located in the upper posterior portion of the right lower lobe and to have increased in size to 4 cm. in diameter. A definite fluid level was present at this time.

In the differential diagnosis, carcinoma, bronchiectasis, and tuberculosis were considered. Bronchoscopic biopsy obtained from the right lower lobe bronchus on Sept. 11, 1936, revealed a well-differentiated squamous-cell carcinoma.

The patient's condition became progressively worse. The temperature ranged between 37 and 38° C. Death occurred on Sept. 22, 1936, after three days of profuse hemoptyses.

At autopsy (Dr. Simon Koletsky) a cavity 6.5 cm. in diameter was found in the upper posterior portion of the right lower lobe (Fig. 17). The cavity wall consisted of friable necrotic tissue. The cavity communicated with the right main bronchus. The walls of the main bronchus, eparterial and hyparterial bronchi were the site of the tumor formation. Tumor tissue extended along the bronchi into the middle and lower lobes. There was direct extension of tumor into the adjacent hilum lymph nodes. Microscopically, the wall of the cavity showed a well-differentiated squamous-cell carcinoma.

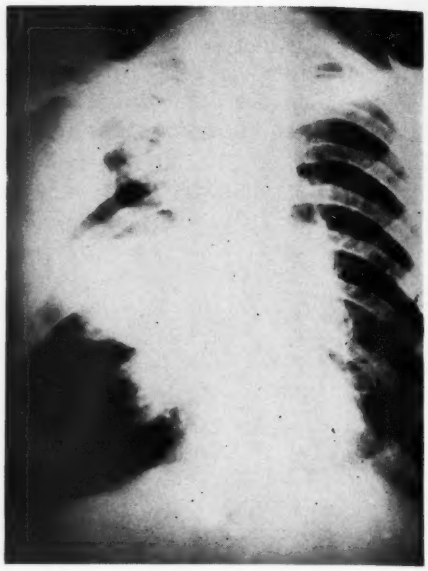

Fig. 18. Case 9. Postero-anterior view of the chest shows a large area of cavitation in the upper half of the right lung. Note the shaggy appearance of the upper portion of the wall. Fluid level in the base of the cavity. This is the largest cavitating carcinoma in this series.

Case 9. J. P., a 64-year-old white male, was admitted on Jan. 14, 1937. The patient had noticed a dry cough for 15 months. Nine weeks before admission it had become severe with production of mucopurulent expectoration. Fatigue, weakness, and loss of 15 pounds in weight had occurred.

There was dullness to percussion over the right upper chest with decreased breath sounds and a marked post-tussive suction. Coarse râles were heard over the entire right lung. The sputum was negative for tubercle bacilli.

Postero-anterior and lateral roentgenograms of the chest made on Jan. 12, 1937, revealed a hugh cavity 14 cm. in diameter in the posterior portion of the right upper lobe (Fig. 18). The cavity contained a fluid level. In the lateral view the cavity was seen to lie somewhat obliquely and to be oval in outline, with its lower pole extending anteriorly and the upper posterior margin at the chest wall. The infiltration forming the wall of the cavity was about 1

cm. in average thickness. The inner border of the upper lateral wall of the cavity was shaggy in outline.

The patient continued to have profuse foul sputum. The lesion was considered to be a lung abscess although the possibility of a bronchogenic carcinoma was also mentioned. On Jan. 18 and 22, 1937, a two-stage drainage of the abscess was performed. Biopsy of the wall showed a moderately well-differentiated squamous-cell carcinoma. After operation the patient failed to improve and died on Feb. 8, 1937. Autopsy was not performed.

SUMMARY

Cavitation occurs frequently in bronchogenic carcinoma resulting from necrosis of tumor tissue, infection, or both. In a series of 127 proven cases, 12 per cent had roentgenologic evidence of cavity formation.

Cavitary bronchogenic carcinomas are often confused with lung abscess and tuberculosis since neither the clinical nor roentgenologic pictures are sufficiently characteristic to establish a conclusive diagnosis.

In the presence of roentgenologic evidence of cavitation, certain findings suggestive of carcinoma may be elicited. Of importance will be the history of an insidious onset, non-productive cough, persistent pain, diminished breath sounds, and bronchographic evidence of bronchial constriction.

Accurate and early diagnosis of cavitary bronchogenic carcinoma depends upon the utilization of the available diagnostic procedures. Of those discussed, bronchoscopic examination is of greatest value, particularly in the differential diagnosis of cavitary pulmonary lesions in patients above the age of 40 years.

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PRIMARY SARCOMA OF THE STOMACH

REPORT OF THREE CASES[1]

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THE stomach is a rare seat of the sarcomas, but they probably are more frequently present than recognized, because of the lack of characteristic clinical symptoms and roentgen diagnostic signs, and the fact that not every gastric tumor, surgically removed, undergoes microscopic study. Even the pathologist may misdiagnose a gastric sarcoma on account of its close resemblance to anaplastic (embryonal) forms of carcinoma (3) and other conditions more frequently found in gastric lesions. Freeman (7) reported a case of gastric sarcoma in which prominent pathologists gave a diagnosis of carcinoma, lymphosarcoma, inflammation, and chronic granuloma.

The first gastric sarcoma was reported by Bruck, in 1847, and the first operation was performed by Virchow, in 1887.

The frequency of gastric sarcoma is about 1 per cent of the malignant gastric conditions, as estimated by Ewing (6), Phillips and Kilgore (14). The ratio was 1 : 121 at the Mayo Clinic (1) during the period from 1908 to 1928. In 4,509 malignant gastric tumors operated upon in seven large clinics in various parts of the world, the sarcoma–carcinoma ratio was found to be 1 : 93. Only one gastric sarcoma has been reported in the Berlin Pathologic Institute (11) among 840 specimens of sarcomas.

The age sector of gastric sarcoma is wider than that of carcinoma; there are cases on record in every decade of life up to the ninth. The extremes are Finlayson's three-and-one-half-year-old and Giacomma's ninety-one-year-old patient. The average age incidence is 43 years, at the Mayo Clinic (1); other reliable statistics state similar age incidence, the most frequent decade being the fourth (9). These data verify the appearance of gastric sarcoma earlier in life than that of carcinoma.

Most of the statistics report an even distribution of cases in both sexes. At the Mayo Clinic, males are affected three times as frequently as females.

Histologically, the gastric sarcomas are classified as fibro-, myo-, lympho-, and myxosarcoma. Fibrosarcoma originates in the submucosa; myosarcoma in the muscularis; lymphosarcoma in the lymph nodes of the submucosa; subserous origin of these sarcomas is less frequent (13).

Clinically, they may be exo-, endogastric, and intramural tumors; well defined or diffuse growths. The exo- and endogastric types are sometimes pedunculated. Fibro- and myosarcoma arise mostly from the curvatures; pyloric involvement is rare. Brodowsky reported a tumor the size of a child's head; Cantwell, one weighing 12 pounds. Also exceedingly small tumors are described. Endogastric growths sometimes undergo ulceration and may cause severe, even fatal, hemorrhages. These tumors usually grow slowly, metastasize late, and form a well-defined mass. Lymphosarcoma involves the pylorus or the curvatures and may produce a diffuse growth but may protrude into the gastric or abdominal cavity as an endo- or exogastric tumor; stenosis has been reported, but it is not common (15). Lymphosarcoma metastasizes early and spreads to the neighboring organs by local extension (8).

The clinical symptoms reported in the literature usually are the same or just slightly deviated from the symptomatology of the carcinoma, but sometimes the addition of clinical symptoms, roentgen signs,

[1] Accepted for publication in June, 1939.

and laboratory findings may be suggestive of a gastric sarcoma.

The general health and condition of the individual is less affected in gastric sarcoma than in carcinoma (2). (It is interesting that we also noticed this to be true in our series of 36 osteogenic sarcomas.)

In some cases the classical symptoms of a gastric or duodenal ulcer are dominated with pain-food-ease rhythm (9).

The general gastric symptoms of malignancy—indigestion, hemorrhage, vomiting—occur later (4) than in carcinoma because the mucosa is only secondarily involved.

Copious hemorrhages with recovery and remissions are a suggestive sign of an ulcerative sarcoma.

Palpable tumors, sometimes huge in size, were present in about 50 per cent of the cases reported by Eusterman and Balfour (5); in some cases they are the leading signs; a large tumor without marked symptoms of malignancy is highly suggestive of sarcoma. The mobility or immobility of the masses has no significance, since freely movable, immobile, and tumors moving with respiration are noted in the case reports.

The blood picture may remain closer to the normal than in carcinoma, although definite secondary anemias have been reported.

The roentgen diagnosis of gastric sarcoma is difficult and is discussed as much as its symptomatology. Golden (8) believes that a well-defined filling defect with a crater shadow in it is suggestive of a sarcoma. He has seen three proven cases of gastric sarcoma with this roentgen finding. Grier (10) states that sarcoma is characterized by a much larger tumor than is carcinoma.

Kandruka and Sierro (5) believe that a pedunculated endo- or exogastric tumor is strongly suggestive of a sarcoma. The pedunculated endogastric tumor is characterized by a sharply defined rounded filling defect with relative mobility and a constant notch at the base of the tumor corresponding to the implantation of the pedicle. The exogastric type produces a narrow pressure defect on the stomach wall at the base of the pedicle with more or less displacement of the organ. Peristalsis may be diminished but not stopped at the attachment, and mucosal folds are not disturbed markedly. These investigators believe that in most cases a lymphosarcoma cannot be differentiated from carcinoma roentgenologically.

Spitzenberger (15) reports a case of lymphosarcoma which originated from the fundus, extended through the diaphragm, and affected the function of the heart. He believes that sarcomas of the upper end of the stomach originate from the greater curvature side of the fundus; carcinomas from the lesser curvature side of the cardia.

Kessler (15) made considerable study of the roentgen appearance of gastric sarcomas and she believes that a flat tumor with smooth borders, a tumor with deeply indented relief, or any single tumor on the greater curvature lacking the characteristic saucer-shape of carcinoma of this location is suggestive of a sarcoma.

The well defined exo- and endogastric sarcomas are best treated surgically—post-operative series of roentgen therapy is quite helpful. The infiltrating types and inoperable cases should be treated by radiation therapy. Extreme caution is required in the dosage of roentgen therapy of gastric sarcoma because of the high radiosensitivity of the tumor. The rapid absorption of the tumor products may cause severe toxicosis, even death. A fatal gastric hemorrhage may occur by the sudden perforation of a larger gastric artery, which was a constant discouraging danger in the pioneer experimentation of roentgen therapy of inoperable gastric carcinoma (12).

To avoid these fatalities, one should divide the area to be treated into small fields and use small daily and total doses. Similar caution should be applied in case of post-operative roentgen treatment of gastric sarcomas.

CASE REPORTS

Case 1. White female, 61 years old, first noticed slight pain in the epigastric region about nine months ago. The pain and discomfort came on about two hours after eating and continued until the next feeding; was easily relieved by food. Occasionally there was gas on the stomach. She had no nausea, vomiting, nor loss of appetite. The patient had lost only a few pounds in weight since first symptoms. She did not notice anything peculiar about her stools, but had had moderate constipation for years. One sister had died of carcinoma; otherwise the family history was not interesting.

Physical Examination.—A poorly nourished, white, elderly female, anemic but not acutely ill. Head and neck essentially negative. Respiration regular, expansion equal; no râles. Heart size and sounds, normal. Blood pressure, 150/100; vessel walls sclerotic. There was a hard, tender, irregular, movable mass in the right epigastrium about seven centimeters in diameter. Extremities and genitalia were normal. Behavior was rational and cooperative. Temperature at admission, 98.2° F.

Laboratory Findings.—Gastric content: mucus, heavy; bile, negative; blood in faint trace; total acid 22, free HCl, 12; red blood cells, 2,980,000; hemoglobin, 58 per cent (Sahli); white blood cells, 8,100; neutrophiles, 84 per cent; blood, Type II; Kline test, negative.

Roentgen Examination.—A stomach of normal size and position was shown. There was a large irregular filling defect at the distal third of the greater curvature, which did not involve the pylorus. Peristalsis was active and stopped at the filling defect. The duodenum was normal. At five hours there was some staining in the stomach and duodenum; the lower ileum was filled. At 24 hours, the transverse and descending colon was filled. Roentgenography of the chest revealed no evidence of metastasis.

A provisional diagnosis of gastric carcinoma was made. The patient received a total amount of 1,600 c.c. citrated blood within a week, which raised the red blood count from 2,980,000 to 4,150,000, and the hemoglobin from 58 to 78 per cent.

On operation, a large growth was found situated in the prepyloric region on the greater curvature. Glands were found in the gastrohepatic and gastrocolic region. The liver, aortic and lumbar glands were negative There was no attachment to the pancreas or colon.

Microscopic Diagnosis.—Small round-cell sarcoma, probably lymphosarcoma.

The patient made an uneventful recovery, was discharged in good condition on the twenty-fourth post-operative day, and was receiving a series of post-operative roentgen-ray therapy.

Case 2. About seven months ago, this patient, a 65-year-old white male, began to have gas on the stomach and epigastric pain which was relieved by belching. This usually occurred after meals, but he was bothered at other times also. Pain was relieved by belladonna several times at the onset of symptoms. There was no noticeable change in his condition until about one month ago, when all his symptoms became more severe and the patient consulted his physician, who prescribed a milk diet. The patient began to vomit immediately after each meal and had the gas and pain as before. There was no blood in the vomitus, which the patient described as tasting neither bitter nor sour, just "awful." His doctor advised him to have an x-ray examination to make sure of the diagnosis. The patient had also complained of constipation for the past seven months, although there was nothing abnormal about the form or color of stool. There was no jaundice. Average weight, 126 pounds; maximum weight, 135 pounds; present weight, 105 pounds.

Physical Examination.—A well developed, poorly nourished, white male, 65 years of age, slightly cachectic. Not acutely ill. No gross abnormalities. Head and neck essentially negative. Chest:

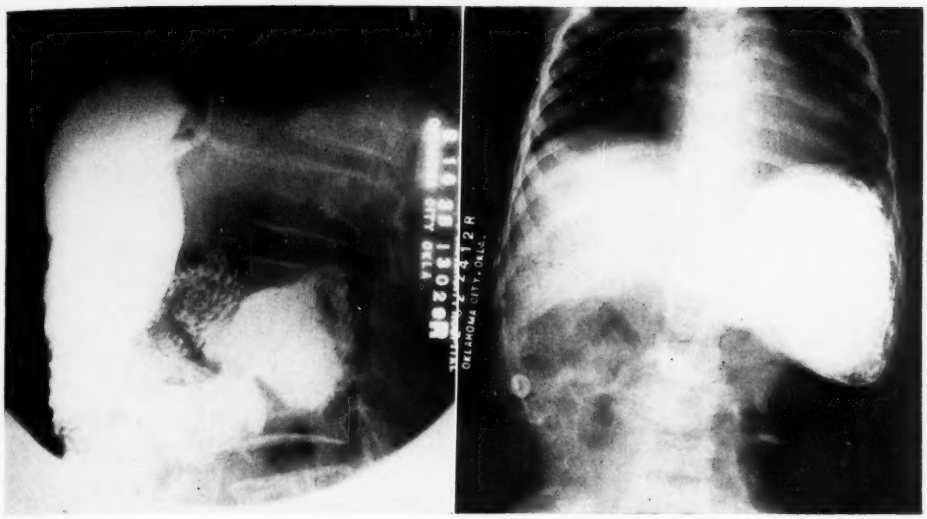

Fig. 1. Case 1. Filling defect with deep indentation on greater curvature.
Fig. 2. Case 3. Constant narrowing of the prepyloric part.

expansion good and equal, breath sounds normal. Heart: blood pressure 126/70; normal in size, shape and position, and rhythm; no abnormal valve sounds. Abdomen: entire abdomen felt soft and doughy except for an indefinitely outlined mass or a spasticity in the epigastrium filling the costal angle; no tenderness. Rectum and genitalia showed nothing remarkable. Extremities: reflexes were sluggish, otherwise, normal.

Roentgen Examination.—Stomach: J-shaped, lower pole was somewhat lower than normal. No filling defects. Peristalsis and duodenal cap were normal. There was a pressure defect on the greater curvature side of the pars media. Slight gastric retention in five hours. Twenty-four- and 48-hour films showed normal advancement of the barium. Diagnosis: Pressure defect caused by an exogastric tumor. The patient was unable to retain barium enema.

Laboratory Findings.—Red blood cells, 5,280,000; hemoglobin, 90 per cent; white blood cells, 11,450; neutrophiles, 84. Gastric content: total acid, 35; free HCl, 15; blood, 0, bile, 0, mucus, moderate. Feces: blood in traces; fat, bile, ova, parasites, negative. Urine, negative. Temperature at admission, 97° F.

Exploratory laparotomy was advised, but the patient refused it, left the hospital, and re-entered four weeks later. The mass now filled the entire upper mid-abdomen, was irregular in outline, semi-fluctuant, tender, and slightly movable. Movement caused pain. Since leaving the hospital, the patient had vomited several times daily, had had black stools, poor appetite, and continued loss of weight and strength. He was now emaciated and willing to submit to operation. Exploratory laparotomy was done on Sept. 22, 1933. On opening the peritoneal cavity, a large hard mass was found in the stomach adherent to the large intestine and pancreas. The patient's condition did not permit further exploration. A small nodule was excised from the greater omentum for biopsy, and the wound was closed in the normal manner. The patient gradually failed and died two days later. Autopsy was not performed.

Microscopic diagnosis of the tissue removed was that of lymphosarcoma.

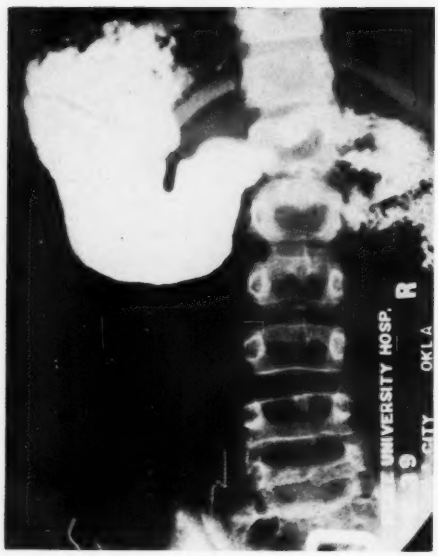

Fig. 3. Case 3. The stomach is normal.

Case 3. This patient, a three-and-one-half-year-old white male, weighed 10.5 pounds at birth. He had lost weight and had not done well on breast milk; had had vomiting spells, at which time he would vomit two days at a time. He had not done well on bottle feeding and had gained very little weight until he began eating the regular diet of a child. The patient had had chicken pox, measles, and whooping cough without complications. He took cold easily. The family history was negative—no accidents nor operation.

The patient had had pneumonia in February, 1932, was ill about one week, made an uneventful recovery but complained of pain in the region of the umbilicus. This pain disappeared after pneumonia but cramping attacks returned two months later, in April. The patient had frequent colds and would complain of pain in the region of the umbilicus associated with each cold. He was always constipated. In November, he had one vomiting spell which lasted four days. There was marked increase in the severity of constipation, with only one bowel action in four days sometimes. He complained of some pain in the kidney regions, and the urine was dark yellow in color. In December, patient began to complain of severe attacks of pain in the abdomen, and he was admitted to the hospital three weeks later.

In the hospital, examination of the abdomen showed some tenderness along the colon and superior to the umbilicus. No masses were palpable. On the second day of hospitalization, severe paroxysmal cramping pain developed in an area just above the umbilicus. This was not relieved by enema, paregoric, or atropine. Patient vomited after onset of pain. Severe vomiting was seen two days later with paroxysmal pain.

Roentgen Examination.—The gastro-intestinal tract revealed a constant narrowing, immediately prepyloric, with extreme gastric retention in five hours and some retention shown on the 24- and 48-hour films. There was also evidence of considerable partial obstruction high in the small intestine indicated by a markedly dilated small bowel loop.

The patient was operated upon the following day for intestinal obstruction. Upon opening the abdomen, a purple-colored distended intestinal mass presented itself in the right upper quadrant and epigastrium. This was gradually manipulated out from the abdomen and found to be a volvulus of jejunum and ileum with about twelve inches of the intestine twisted. When the volvulus was untwisted and the adhesions broken up, the mesentery was found to contain many discrete hard tumors from 0.5 to 1 in. in diameter. There were some clusters of these tumors and, apparently, their weight had caused the volvulus. One of these tumors was removed for biopsy. The patient made an uneventful recovery.

Microscopic Examination.—The node removed showed a lymphoblastic process, apparently superimposed on some low-grade chronic type of inflammation. Microscopic diagnosis: lymphoblastoma, probably lymphosarcoma and chronic lymphadenitis.

Fourteen days after operation, a course of roentgen therapy was administered to the upper abdomen. Three months later, the patient returned for a check-up examination, was practically symptomless, but roentgen examination of the gastro-intestinal tract still revealed a constant rigid narrowing of the prepyloric part; five-hour film showed slight gastric retention, otherwise normal advancement of the barium; 24-hour film showed barium to be distributed in the colon. The previous roentgen treatment was repeated. Since the second course of roentgen therapy, the patient has remained symptomless, developed normally, and the last roentgen examination, done six and one-half years after the first irradiation, revealed a stomach normal in shape, size, and position, with mucosal folds, peristalsis, and duodenal cap regular. At five hours, there was no gastric retention, the lower ileum was filled with the barium; at 24 hours the barium was distributed in the colon.

Summary of Case Reports.—Three cases were reported from our series of gastro-intestinal sarcomas, in which the diagnosis and localization of the primary lesion were verified by microscopic and x-ray examinations and also by surgical exploration. The cases in which the diagnosis or origin of primary lesion was doubtful are not mentioned in this report.

One of our patients is probably the youngest individual (three and one-half years old) having gastric sarcoma to have been reported in the literature; the other two are in the sixth decade.

Case 1 had typical peptic ulcer symptoms with pain-food-ease rhythm of nine months' duration, without nausea or vomiting. She had a large endogastric tumor with deeply indented relief, indicating destruction of the mucous membranes, resulting in occult bleeding, anacidity, and anemia.

Case 2 had fairly typical ulcer symptoms of nine months' duration with nausea and vomiting in the last month. Since his tumor was exogastric, the gastric mucosa was not involved, consequently the blood picture was normal, gastric acidity only slightly lowered, occult bleeding only in traces.

Case 3, we believe, is one of the youngest patients having gastric sarcoma reported in the literature. Although a diagnosis of sarcoma of the stomach was made by roentgen examination alone, we admit that the age of the patient was of considerable importance in differentiating it from carcinoma. Summing up the history of unusual frequency of vomiting, indigestion starting soon after birth, constancy of gastric symptoms, disseminated metastasis at three and one-half years of age, we believe this is a case of congenital lymphosarcoma of infiltrating type.

Conclusion.—Pre-operative diagnosis of a gastric sarcoma is extremely difficult, since there are no characteristic symptoms and signs to differentiate it from carcinoma.

The age incidence of gastric sarcoma is earlier than that of carcinoma, a gastric tumor in the first three decades of life being highly suggestive of a sarcoma. The duration of symptoms is usually shorter than that of carcinoma.

A large tumor with diminutive symptoms of malignancy is strongly suggestive of a sarcoma. Gastric sarcoma may simulate a peptic ulcer symptomatically.

Hemorrhage with recovery and recurrence without marked anemia is a suggestive sign. Loss of weight is noted less frequently in sarcoma than in carcinoma.

Roentgenologically, a flat tumor with smooth borders, pedunculated growth, isolated filling defect on the greater curvature with deep indention or with a crater shadow in it, and tumors on the greater curvature side of the fundus, are suggestive of sarcoma. Due to the radiosensitivity of sarcoma, an inoperable tumor may be rendered operable after a course of roentgen therapy and in some instances the tumors are curable by radiation alone.

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RESPONSES OF *DROSOPHILA* PUPAE TO X-RAYS[1,2]

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From the Biophysical Laboratory, Memorial Hospital

EGGS of *Drosophila melanogaster* have been found by a number of investigators, particularly Packard (1), to possess properties which make them favorable as test objects for radiobiologic investigations. They are small, comparatively radiosensitive, usually easily obtainable, and manifest a clear-cut response to radiation (hatching or failure to hatch). However, as pointed out by this laboratory (Henshaw and Henshaw, 2), and by Packard (3), a marked variation in radiosensitivity occurs with age, in these forms, and it is difficult, if not impossible, to control the age factor adequately in experiments involving collection and treatment intervals of more than a few minutes.

Mavor (4), in 1923, pointed out that *Drosophila* pupæ have a period of several hours during which their radiosensitivity is nearly constant. Since we had need for new test material for quantitative radiobiologic work, experiments were carried out to determine the suitability of these organisms. At the outset we may say that they were found suitable and that a paper from this laboratory has been published already, based on work involving their use (Müller, 5). Although several times larger than eggs, the pupæ are, nevertheless, small and convenient to handle, are easily obtainable, resistant to drying, uniformly sensitive to radiation for a number of hours, and, like the eggs, give a clear-cut response to radiation (*i.e.*, they either emerge or fail to emerge as imagos). It is the purpose of this report to present the ground work experiments which justified the use of this organism as test material as well as to present some of the interesting radiobiologic relationships which have been observed.

The following will be dealt with particularly: (1) a description of pupal (and pre-pupal) development as related to the problem at hand, including methods of collecting, restricting age, and identifying stages of development; (2) changes in radiosensitivity with age; (3) the relation between radiosensitivity and developmental activity; (4) reproducibility of the quantitative response of pupæ to x-rays, and (5) the influence of lethal doses of x-rays on respiratory rate.

1. *Pupal Development.*—Starting with the freshly fertilized egg of *Drosophila*, the adult stage is reached in about ten days when maintained at 26° C. The egg develops along lines typical of insects, in general, and hatches into a larva in from 18 to 24 hours. The larva is a moving form which feeds actively and grows continuously. After about five days of larval activity, the organism ceases feeding and crawls up the sides of the culture bottle in preparation for becoming a pupa. After crawling about for several hours, movement begins to decrease and finally stops altogether. As activity ceases, the oral hooks are withdrawn, the anterior spiracles are everted, and a thin chitinous case, or *puparium*, is secreted about the organism.

Since the present investigation was first undertaken, a detailed embryologic account of pupal (and pre-pupal) development of *Drosophila melanogaster* has been published by Robertson (6). The reader is referred to this for additional information. Robertson has pointed out, with justification, that in order for *Drosophila* development to be consistent with that established for other insects, true pupation does not occur until about 11½ hours after cessation of movement. However, since this technical embryologic consideration does not concern us here and since we were interested in the complete quies-

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[2] Accepted for publication in May, 1939.

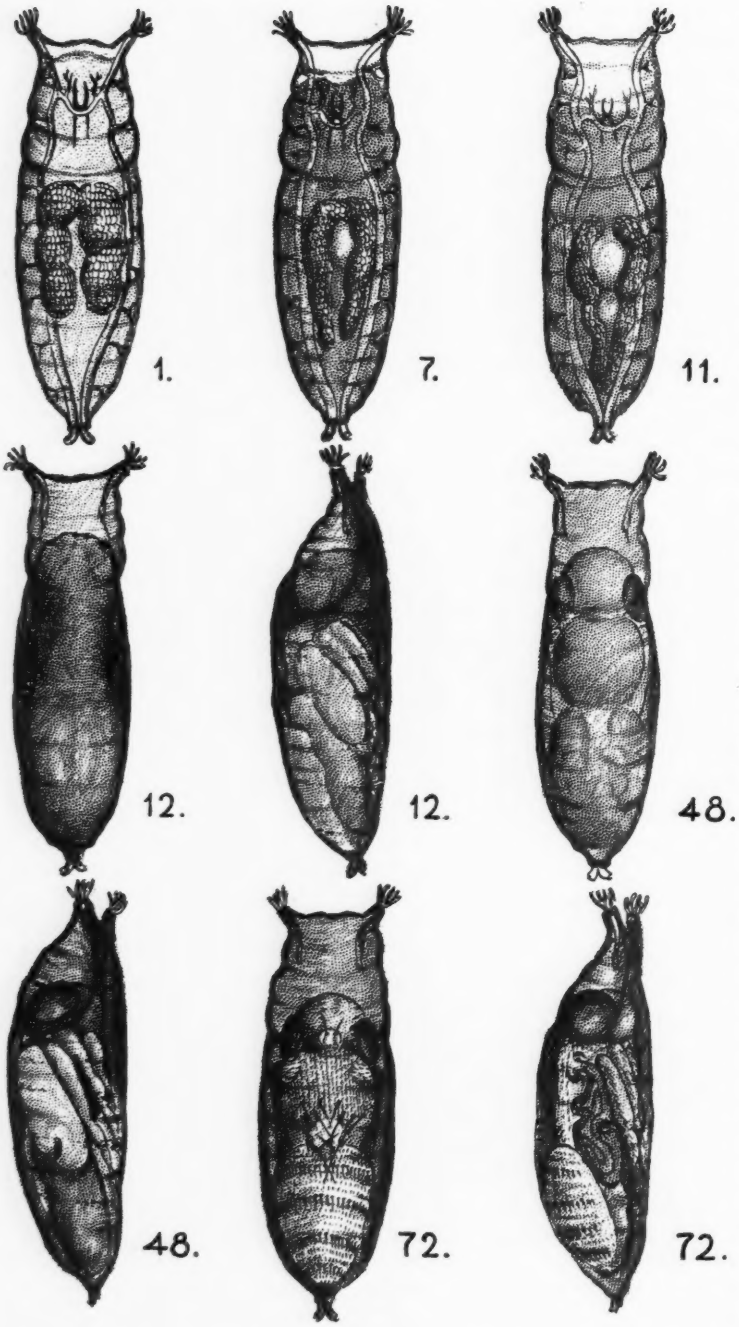

Fig. 1. Drawings showing stages during pupal (and prepupal) development which may be easily recognized with the unaided eye. The numbers indicate the age in hours after the collection of immotile white forms (*i.e.*, when maintained at 26° C.).

cent period, we shall, as a matter of convenience, refer to the cessation of movement as the beginning of pupation. All subsequent mention of pupal age, therefore, will be with reference to this stage as zero, unless otherwise indicated.

When the puparium is first formed it is white and translucent (Fig. 1, 1). Externally, the spiracles, two anterior and two posterior, are most conspicuous. Those in front are wide apart and branched, whereas those behind are close together and not branched. The anterior and posterior spiracles are connected through the body by two hollow trunks which are rendered clearly visible to the unaided eye by a combination of reflected and transmitted light. Beside the trunks, there are two other organs plainly visible within—the oral hooks (completely withdrawn) at the anterior end and the salivary glands located in the central region.

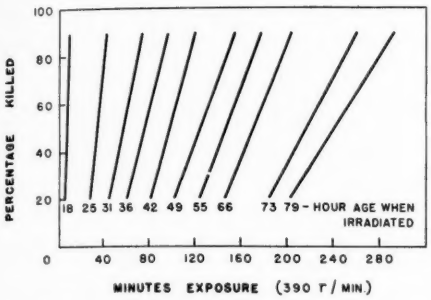

Fig. 2. Graph showing the percentage (20 to 90) of organisms killed as a function of exposure to radiation when treated at different ages. The curves for 2-, 6-, and 12-hour material were identical with the one for 18-hour material and are, therefore, not shown.

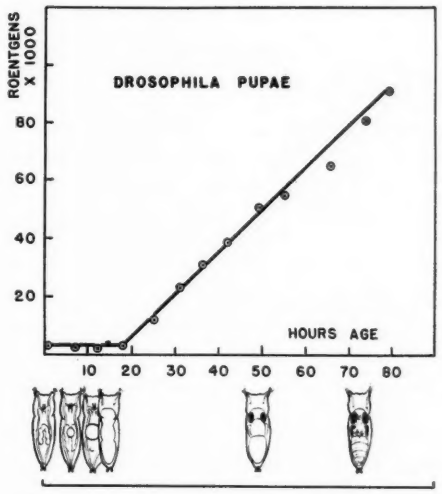

Fig. 3. Curve showing the number of roentgens (measured in air) required to kill 50 per cent of the organisms in a sample at different stages in development. The diagrams beneath show the distinguishing features at different stages.

Within two hours after the cessation of movement, the organisms begin to turn brown and more opaque, a change which becomes complete within four hours. When the puparium is first formed, it is firmly attached to the tissues and organs beneath, but within three hours the structures in the anterior region begin to retract, causing a gas chamber to be formed. At about the same time, a second gas chamber appears in the central region. As these enlarge, the specific gravity of the organisms decreases so that they change from sinking to floating forms (Fig. 1, 7) at about seven hours, as determined by placing them in a vessel of water. Enlargement of the central gas chamber continues until its diameter occupies about 80 per cent of that of the entire organism. This stage is reached at about 11 hours (Fig. 1, 11).

Although the appendage primordia and the head anlage are well established at 11 hours, the organism appears headless and ill defined. However, within a 15-minute period beginning at approximately 11½ hours, it changes into a form with clearly defined head and body regions and with appendage rudiments. This takes place mainly through the collapse of the central gas chamber, eversion of the head and appendage primordia, and constrictions which set off the head, thorax, and abdomen (Fig. 1, 12).

After the elaboration of body parts at 11½ to 12 hours, no other externally visible change occurs until the organisms reach 48 hours of age (Fig. 1, 48). At this time red pigment begins to show in the eyes, the color becoming more intense during the following hours. The next distinctive change occurs at 72 hours when black pigment appears in the bristles, legs, and wings (Fig. 1, 72). Emergence of the imago from the puparium occurs at 96 hours.

There are, therefore, a number of easily recognizable stages manifest during the pupal period which may be used to designate the degree of development. These, together with the approximate times of occurrence (*i.e.*, when maintained at 26° C.), may be listed as follows:

	STAGE	HOURS
1.	Motility ceases	0
2.	Browning begins	2
3.	Organism will float on water	7
4.	Delimitation of body regions	11½
5.	Appearance of red eye pigment	48
6.	Appearance of black pigment in bristles, legs, and wings	72
7.	Emergence	96

2. *Changes in Radiosensitivity with Age.*—Investigation of the changes in radiosensitivity with age was undertaken with two points in mind: (1) determina-

tion of the suitability of *Drosophila* pupæ as test material for quantitative radiobiologic work, and (2) correlation of the changes in susceptibility to radiation with changes in the character of the developmental activity. Only the first point, however, will be dealt with in this section.

In order to correlate radiosensitivity and age in a significant way, it was necessary to restrict the age of individuals in the samples at various stages to fairly narrow limits. This was done by making use of the information given in Section 1 that the organisms are white for only about two hours after motility ceases. The procedure, therefore, was to collect immotile white forms and keep them in moist chambers at 26° C. until they reached the stage to be investigated.

Since the organisms are immotile and white for such a small portion of their life span, only a few will be found in this stage even in heavily populated cultures, thus making collection a real task. For this reason, the first experiments were performed with only two organisms per sample. By this means it was possible to ascertain the approximate radiosensitivity at different stages with a minimum of effort.

The samples were prepared by removing white pupæ from the walls of culture bottles with a soft wet brush and placing these on small squares of moist filter paper. When they had reached the age to be tested, the samples were arranged on a thin celluloid tray and taken to the x-ray machine for treatment.

The x-ray equipment used consisted of a Coolidge type, tungsten target, water-cooled tube operated at 200 kv. and 30 ma. At 33 cm. distance from the center of the target and with no filter, this equipment delivered 390 r/min. (measured in air with a small spherical celluloid ionization chamber).

By using emergence as an end point, it was soon found that for the first 18 to 20 hours of pupal life the radiosensitivity changes little, if any, but that following this period it increases continuously until the end of development—thus confirming, in a general way, the observations of Mavor.

Having thus obtained an impression of the doses of radiation required at the different stages, more precise experiments were designed. The improvements, in the main, consisted of an increase in the number of organisms per sample and a clearer definition of the end point. Instead of two organisms per sample being used as before, 50 were employed and the killing as an end point was restricted to death at a particular stage. In case of the preliminary experiments, it was found that most of the pupæ which failed to emerge as imagos developed to the point of having well-defined red eyes and black bristles, legs, and wings. Thus, from the chart in Section 1 it appeared that development was fairly normal up to the point of emergence; it seemed as though the organs having to do with emergence had been injured or destroyed by the radiation (perhaps the glands which secrete the enzyme that weakens the puparium prior to the escape of the imago). Interestingly, this happens irrespective of when the pupa is irradiated in its life cycle (and even when irradiated during the larval stage).

Occasionally, however, in the preliminary experiments, it was found that some of the pupæ which failed to hatch did not display pigment and were of an even yellow color, thus indicating that death had occurred at an earlier stage in development. Investigation of this disclosed that if the dose of radiation is adequate, the pupæ may be killed at earlier stages, and, further, that usually about 1 or 2 per cent of the organisms in a sample die at the early stage whether irradiated or not. Thus, since we wished to obtain as uniform a response to the radiation as possible, it was decided to exclude arbitrarily from consideration all organisms which did not develop to the stage of black pigment in bristles, legs, and wings. This greatly improved the regularity of the experimental data because it restricted the ef-

fective dosage to what amounted to a threshold killing dose.

Four experiments were performed on material at the following ages: 2, 6, 12, 18, 25, 31, 36, 42, 49, 55, 66, 73, and 79 hours (*i.e.*, from the time white immotile pupæ were collected).

The data obtained were plotted to show how the percentage of organisms killed varied with the dose of radiation applied. It was thus found that for the range between 20 and 90 per cent killing, the experimental points could be fitted satisfactorily with straight line curves. The curves obtained in this manner for various ages of material and based on four separate experiments are shown in Figure 2. (The curve for 18-hour material also represents that for 2, 6, and 12 hours since the results obtained were identical in all these cases.)

It will be seen that beyond 18 hours the threshold effective dose becomes larger and that the range between the threshold and complete killing dose is increased. In order to obtain a clearer idea of how the changes in radiosensitivity occur, values were read from the curves at the 50 per cent level and plotted as shown in Figure 3. In this figure the curve appears to be flat for the first 18 hours, at which point it abruptly turns upward at an angle and again follows a straight line course. This means that the organisms do not change in susceptibility appreciably during the first 18 hours of pupal development, but that beyond this age they increase steadily in resistance. The significance of this interesting curve will be dealt with in the next section.

3. *Relation between Radiosensitivity and Developmental Activity.*—Having determined the changes in radiosensitivity with age, it became of interest radiobiologically to correlate the changes in susceptibility with the varied developmental activities at the different stages in order to ascertain whether or not any significant relation existed between them.

Since the larva is essentially a feeding form, it elaborates an extensive musculature for movement, highly specialized alimentary tract and gland system for the assimilation of food, and a complicated tracheal system for respiration. Thus, since, in general, these are of little or no use to the pupa or mature fly, one of the first functions of the organism following puparium formation is the removal of many of the larval structures. This takes place during the first 18 to 24 hours, mainly by histolysis, phagocytosis, and the casting off of structures. These functions constitute the major portion of the developmental activity during this period. The larval musculature disappears almost completely, the main portion of the digestive system is replaced, and the tracheal system is greatly modified.

From 24 to 48 hours, the organism is in a stage of relative inactivity. During this period there is little destructive activity and likewise little organogenesis. Active organogenesis, however, begins at about 48 hours and proceeds at a rapid rate until the imago emerges.

Three quite distinct stages are, therefore, recognized: the first, from 0 to 24 hours, which is characterized largely by destructive changes; the second, from 24 to 48 hours, by quiescence, and the third from 48 to 96 hours, by constructive changes.

In order to obtain a more tangible idea of the growth activity and at the same time some quantitative information on the developmental activity, it was decided to measure the rate of oxygen uptake at the different stages. For this a Warburg type of microrespirometer was used.

Samples consisting of 10 white pupæ each were placed in several different vessels and determinations were made daily. Each daily determination consisted of a series of four readings taken at 30-minute intervals. Since we were concerned only with relative respiratory rates at the different ages rather than the actual amounts of oxygen consumed and since the volumes of the vessels used varied less than 2 per cent, our results have been expressed in terms of millimeters change

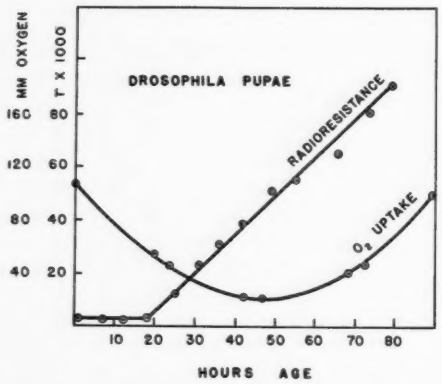

Fig. 4. Curves showing the relationship between oxygen uptake and radioresistance at different ages (pupæ only).

problem under consideration. The averaged values are plotted together with the results for changes in radiosensitivity (Fig. 4).

It will be seen that the rate of oxygen consumption drops gradually during the first 40 to 50 hours, after which it rises again. Because of the distinct downward trend of the curve for oxygen consumption at the beginning, it seemed pertinent to study its course during larval development. Accordingly, it was decided to extend the curves for both oxygen consumption and radiosensitivity over the entire life cycle of *Drosophila*. This was done in accord with methods already

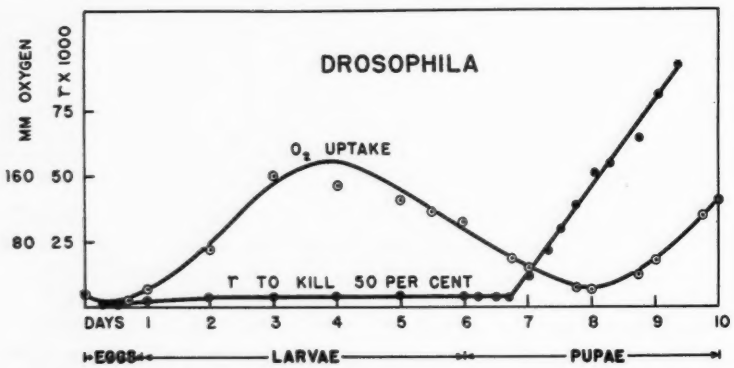

Fig. 5. Curves showing the relationship between oxygen uptake and radioresistance at different ages (entire life cycle).

in meniscus level of Brodie's fluid in the manometers, the readings, of course, receiving the customary thermobarometric corrections.

The values for changes in level of meniscus were plotted as a function of time and the best straight line drawn among the points. Readings were then taken from the curve for a period of 75 minutes respiration, this interval being chosen arbitrarily but always kept the same. After a series of exploratory tests, four such experiments were carried out and the results averaged. No table of values is being presented since a statistical evaluation of the data is not essential for the described and the results are given in Figure 5.

Figure 5 shows that the radiosensitivity changes little during larval development and that it is essentially the same as that for the early pupal stage. The rate of oxygen uptake, on the other hand, follows a varied course which may be correlated in a general way with the developmental activity. As the larva feeds and increases in size, its respiratory rate per organism increases, but with the approach of larval quiescence, this rate begins to fall. During the late larval and the early pupal stages, the fall continues at a rapid rate. However, when the destructive

changes slow down, the decline in rate of oxygen uptake likewise tapers off and as active organogenesis sets in there is a corresponding increase in oxygen uptake. The curve showing the rate of oxygen uptake may, therefore, be taken as a graphic representation of the developmental activity.

Examination of the two curves (Figs. 4 and 5) shows what appears to be a complete lack of positive correlation between rate of oxygen uptake and radiosensitivity.

4. *Reproducibility of Quantitative Responses to Radiation.*—Since we are concerned in this report with indicating the reliability of *Drosophila* pupæ as test material for radiobiologic work, it is necessary to indicate the precision of results obtainable.

It was indicated above in Section 2 and in Figures 4 and 5 that the radiosensitivity does not change appreciably during the first 18 hours of pupal life and that the susceptibility remains quite constant during this time. This long period of unchanging susceptibility appeared especially favorable for various types of experimentation and was accordingly investigated for precision of response.

The procedure used to limit the age of test material for this purpose was as follows: Active cultures of flies were maintained by seeding several bottles daily. To collect young pupæ, several such culture bottles containing many larvæ and a few pupæ were selected and the inner walls scraped free of pupæ at about 5 P.M. These bottles were then placed in the incubator overnight to allow other larvæ to crawl up the sides and change into pupæ. At 9 A.M. the newly formed pupæ were carefully removed with a soft wet brush and placed in a small vessel of water. Gentle stirring washed food particles free and allowed the larvæ and a few of the youngest pupæ to settle to the bottom. The floating pupæ were then removed to small moistened filter paper squares in a moist chamber and arranged side by side in rows (the latter to facilitate counting). The age limit of test material at the time of collection was, therefore, 7 to 16 hours since floating begins at seven hours (Section 1) and since the interval from 5 P.M. to 9 A.M. is 16 hours. There was no important reason for eliminating the pupæ less than seven hours old but these organisms dropped out in the washing procedure employed, and, since material was plentiful, no attempt was made to recover them. The samples as prepared for treatment consisted of 50 pupæ each.

Irradiation, which was carried out under precisely the same conditions as described in Section 2, was completed within one hour following collection and, therefore, occurred safely within the period of consistent radiosensitivity. Determination of the effect was, likewise, performed as described in Section 2.

The results for 17 consecutive experiments are shown in Table I. The values given represent the percentage killed in each sample of 50. Averages are shown at the bottom of each column together with the Standard Error of the Means. The averages and the limits of error have been indicated in the graph (Fig. 6).

TABLE I.—PERCENTAGE KILLED

Min. Expo.	0	5	6	7	8	9	10
1	2	7	26	63	94	100	100
2	0	8	20	62	82	100	100
3	0	10	21	28	71	100	100
4	0	1	19	38	78	95	100
5	2	10	27	52	89	100	100
6	4	12	21	43	77	98	100
7	0	6	16	47	71	100	100
8	0	3	26	44	75	95	100
9	3	10	21	46	83	99	100
10	3	12	15	52	86	97	100
11	2	11	18	36	59	95	100
12	3	13	28	57	88	98	98
13	2	7	32	52	84	100	100
14	5	8	35	41	86	95	98
15	4	12	33	50	70	98	100
16	0	14	46	58	90	98	100
17	5	12	29	67	85	100	100
Av.	2.6	9.2	25.5	49.2	80.9	98.1	100.0
σ_m	0.47	0.84	1.88	2.45	2.06	0.44	0.05

Examination of Figure 6 discloses (1) that the fertility of the controls is high; (2) that the reproducibility of the results is within limits satisfactory for quantita-

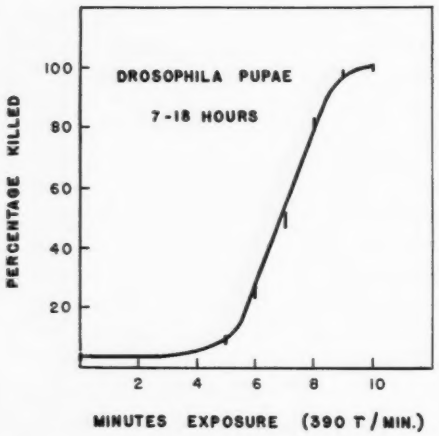

Fig. 6. Curve showing the percentage killed as a function of exposure to radiation, the vertical distance of the plotted points indicating the standard error of the mean.

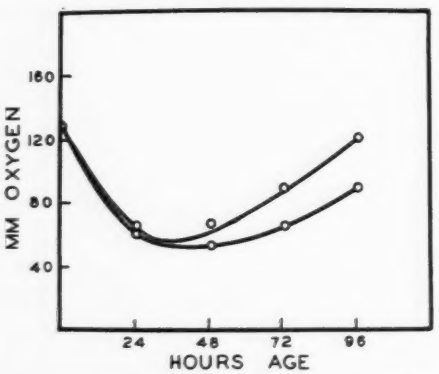

Fig. 7. Curves showing the rate of oxygen uptake during the pupal period for non-irradiated material and for material which received a lethal dose of radiation (5,000 r) when at the stage of white pupæ.

tive work; (3) that a distinct threshold effect is manifest, and (4) that the curve is of the sigmoid type.[3]

5. *Influence of a Lethal Dose of X-rays on Respiratory Rate.*—As a supplement to the measurements of respiratory rate in Section 3, similar measurements were made on organisms which had received lethal doses of radiation. This was done in an attempt to ascertain whether or not the respiratory activity is disturbed during development when a lethal dose of radiation is given early in the pupal period and death occurs late in this period.

For this work the dose of 5,000 r was used, throughout. This dosage was chosen for two reasons: first, because it is safely beyond the dose required to prevent 100 per cent of the organisms in a sample from hatching (Fig. 6), and second, because it was found in exploratory experiments that this dose does not prevent development from reaching the stage of black pigmented bristles, legs, and wings (72 hours, Section 1). The treatment, therefore, produced the same end points dealt with above and at the same time insured 100 per cent lethal effects in the samples.

The procedure used in collecting material, measuring the rate of oxygen uptake, and analyzing the results was the same in all essential details as described in Section 3. Six separate experiments were carried out and the averaged results have been plotted in Figure 6.

It will be seen that so far as these data are concerned the rate of oxygen uptake remains about the same during the first 20 to 30 hours but that it drops below and behind that of the non-irradiated material beyond this point.

It would appear from this that the radiation does, to some extent, affect the respiratory activity. Although completely satisfactory proof cannot be brought forth to the contrary, two objections may be raised at the outset against such an interpretation. First, it was observed in connection with the work described above (Sections 2 and 3 in which sub-lethal doses were administered) that emergence of the imago was delayed by several hours (6 to 10 in some cases). Going farther, it is reasonable to expect that for larger doses even greater delay would be produced. In accordance with this, it may be pointed out that the level of respiratory rate

[3] It will be noted that the curve presented here indicates that the organisms are somewhat more sensitive than does Müller's curve. This is because Müller's organisms varied in age from seven to 24 hours, his experiments being carried out before the radio-sensitivity-age factor was fully understood.

reached by the controls, say at 38 hours, is not reached by the irradiated until 64 hours—a delay of 22 hours; or that the level reached by the controls at 72 hours is not reached by the irradiated until 96 hours—a delay of 24 hours. From this it would seem that the delay in progress of the respiratory activity might be attributed to slower development.

Second, in addition to delay in attainment of levels of respiratory activity, the rate for the irradiated forms drops to a lower minimum at from 40 to 50 hours. This drop indicates that the respiratory rate per individual after receiving the lethal dose of radiation actually decreases. One cannot say, however, that this is due to the action of radiation on the respiratory apparatus since the findings thus far do not yield information as to the number of cells functional in the two cases at corresponding ages. Such information is needed before conclusions can be drawn.

The results presented here, therefore, lead to the presumption that although the respiratory activity may be altered somewhat, it is not by this means that the radiation exerts its lethal influence.

SUMMARY

1. The use of *Drosophila* pupæ as test objects for quantitative radiobiological work has been considered and investigated. Further, as a part of this investigation and as a supplement to it, the relation between radiosensitivity, developmental activity, and respiratory rate at various stages during the pupal period has been determined.

2. Using as a criterion of effect, development to the stage of pigmented bristles, legs, and wings, and failure of the imago to emerge from the puparium, it was found that during the first 18 hours the organisms maintain a strikingly uniform radiosensitivity, but that beyond this point their resistance increases at a rapid rate.

3. Considering normal development alone, three fairly distinct phases were recognized: the first, from 0 to 24 hours, characterized mainly by destruction and removal of larval organs; the second, from 24 to 48 hours, by relative quiescence, and third, 48 to 96 hours, by active organogenesis.

4. It was found that the rate of oxygen uptake in these forms decreases during the first phase, reaches a minimum during the second, and increases during the third, reaching a level at the time of emergence about equal to that at the beginning of the pupal stage.

5. There was, therefore, a positive correlation between the rate of oxygen uptake and developmental activity, but none between either of these and radiosensitivity.

6. The reproducibility of the quantitative response of pupæ 7 to 18 hours old to radiation was tested and found to be within narrow limits.

7. The general shape of the curve showing rate of oxygen uptake during the pupal period was not changed appreciably when lethal doses of radiation were administered early in the period. Only a slight delay was manifest which probably has little significance.

CONCLUSIONS

Drosophila pupæ may be recommended as favorable test material for radiobiologic studies for the following reasons: (1) they are easy to obtain in all seasons; (2) they are small, having a diameter of about 1 mm. and a length of about 2.5 mm.; (3) they are resistant to drying; (4) their susceptibility to radiation remains nearly constant for a period of about 18 hours; (5) they are relatively radiosensitive, 50 per cent being killed by a dose of 2,700 r, and (6) they yield results which can be reproduced within narrow limits from day to day.

Acknowledgments.—The authors wish to express their appreciation to Dr. G. Failla for his interest and suggestions in connection with this work, to Dr. M. D. Schweitzer for carrying out some of the preliminary experiments which lead to the

ones presented here, and to Mr. J. C. Bender for part of the drawings.

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ROENTGENOGRAPHIC UNSHARPNESS OF THE SHADOW OF A MOVING OBJECT—II[1,2]

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INTRODUCTION

IT IS the purpose of this paper to present certain analytic and experimental results which have been obtained since the first paper[1] on this subject was published. These results are discussed briefly in three sections:

V.[3] Additional analytic data of the variation of exposure with distance along the unsharp shadow border when the intensity of the x-ray beam is constant.

VI. Tests by Dr. G. S. Pesquera[4] of the observed unsharpness of the shadow of a moving plane object; the technics for these tests were equivalent to modern chest roentgenographic technics.

VII. Analytic and experimental results for a moving plane object with an x-ray beam the intensity of which varies exponentially with time. The test films were made by R. B. Wilsey.[5]

A short description of the symbols used in this paper and in the first paper[1] follows. Figure 1 shows the arrangement of the film F, the moving plane object O, and the *effective* focal spot f; linear dimensions are in centimeters. The following quantities are used in the analysis; the x's are measured from x_0 upward:

$$x_1 = \frac{bf}{a} \qquad x_2 = \frac{a+b}{a}\,vT$$

$$vt_1 = \frac{bf}{a+b} \qquad x_3 - x_2 = x_1 = \frac{bf}{a}.$$

a = distance (cm.) from focal spot to object
b = distance (cm.) from object to film
f = length (cm.) of one side of square effective focal spot
v = velocity (cm./sec.) of object edge, parallel to film
T = exposure time (sec.)
x_1 = geometric unsharpness (cm.)
x_2 = unsharpness due to motion (cm.)
x_3 = "total unsharpness" (cm.).

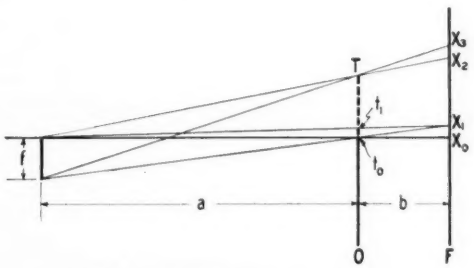

Fig. 1.[6] Schematic diagram *not drawn to scale* of the trace f of the effective focal spot of a roentgen-ray tube, the trace O of a plane object which moves with constant velocity v cm. per second in the direction t_0T, and the trace F of a plane x-ray film. A perspective view of the experimental apparatus is shown in Figure 5 of the first paper.[1]

The exposure, E, at any point on the film was calculated by integrating the instantaneous x-ray intensity at that point from $t = 0$ to $t = T$. The intensity of the beam at points outside of the shadow of the object was called I; the intensity of the beam at points *within* the shadow of the object was called I_0. Expressions for the exposure as a function of x were calculated

* The Laboratory is organized under grants from Lessing J., and William, Rosenwald, the University of Pennsylvania, and the National Tuberculosis Association.

[1] The first paper with this title was published in RADIOLOGY, **28**, 450–465, April, 1937.

[2] Accepted for publication in August, 1939.

[3] The last section of the paper listed in Footnote 1 is numbered IV.

[4] Of the Metropolitan Life Insurance Company Sanatorium, Mt. McGregor, N. Y.

[5] Of the Research Laboratories of the Eastman Kodak Company, Rochester, N. Y.

[6] This figure is from the first paper.

(α, β, δ are values of x within ranges defined below):

$$E_\alpha = \frac{a^2(I - I_0)}{2bfv(a + b)}\alpha^2 + I_0T \qquad \left(0 \leqslant \alpha \leqslant \frac{bf}{a}\right)$$

$$E_\beta = \frac{a(I - I_0)}{v(a + b)}\beta + \frac{bf(I - I_0)}{2v(a + b)} + I_0T \qquad (0 \leqslant \beta \leqslant x_2 - x_1)$$

$$E_\delta = -\frac{a^2(I - I_0)}{2bfv(a + b)}\delta^2 + \frac{a(I - I_0)}{v(a + b)}\delta - \frac{bf(I - I_0)}{2v(a + b)} + IT \qquad (0 \leqslant \delta \leqslant x_3 - x_2).$$

V. ADDITIONAL ANALYTIC DATA FOR CONSTANT INTENSITY X-RAY BEAM

The formulas listed above for E_α, E_β, and E_δ were derived in terms of the arrangement shown in Figure 1. In this figure the object edge is at t_0 at the beginning of the exposure; the object extends *downward* from t_0 at this instant; the object moves upward during the exposure. In the experimental apparatus (see Fig. 5 of first paper[1]) the object edge is at t_0 at the beginning of the exposure; the object extends *upward* from t_0 at this instant; the object moves upward during the exposure. No explicit proof was given in the first paper that these two arrangements produce the same results. Such a proof follows.

When the object O is *below* t_0 at the beginning of the exposure, the exposure at any point from x_0 to x_3 may be calculated by means of one of the three expressions for E_α, E_β, E_δ given above. When the object is *above* t_0 at the beginning of the exposure (as it was in the experimental set-up) the exposure from $x = x_0$ to $x = x_1$ may be obtained by changing the expression for E_α in two ways:

1. Interchange I and I_0, because the object covers those parts of the film formerly uncovered, and *vice versa*.
2. Make the substitution $\alpha = -\left(\delta' - \frac{bf}{a}\right)$, because the range of α in the first case is the low density edge of the shadow; in the case now under consideration the x_0 edge of the unsharpness is the high density edge; finally, this transformation of α to a new variable δ' provides that δ' be zero at x_1 and $\frac{bf}{a}$ at x_0.

The result obtained by means of these operations may be written:

E_α (with I and I_0 interchanged, and with α replaced by $-\left(\delta' - \frac{bf}{a}\right)$

$$= -\frac{a^2(I - I_0)}{2bfv(a + b)}\delta'^2 + \frac{a(I - I_0)}{v(a + b)}\delta' - \frac{bf(I - I_0)}{2v(a + b)} + IT \qquad \left(0 \leqslant \delta' \leqslant \frac{bf}{a}\right)$$

This is precisely the expression for E_δ. Therefore, the values of E from x_2 to x_3 when the object starts from a position *below* the t_0 line are equal to the values of E from x_1 to x_0 when the object starts from a position *above* the t_0 line. Similar transformations show that: (1) The values of E from x_1 to x_2 when the object starts from a position *below* the t_0 line are equal to the values of E from x_2 to x_1 when the object starts from a position *above* the t_0 line and, (2) the values of E from x_0 to x_1 when the object starts from a position *below* the t_0 line are equal to the values of E from x_3 to x_2 when the object starts from a position *above* the t_0 line.

Therefore, the curves of exposure *versus* x measured from the less dense side of the unsharpness are the same, whether the object starts below the t_0 line and moves upward, or the object starts above the t_0 line and moves upward. The unsharpnesses of the two edges of an absorbing strip which moves across a film will be mirror images of each other; the curve of density *versus* x measured from the location of the mid-point of the object at time $T/2$ will be symmetrical about the line

TABLE I

Roentgen-ray Tube Voltage (kv. p.)	Roentgen-ray Tube Current (ma.)	Exposure Time (sec.)	Focal Spot-film Distance (ft.)	Side of Square Effective Focal Spot (mm.)	Velocity of Object for which Technic Produces Minimum Unsharpness (cm.-sec.)
70	150	1/10	5.5	1	1
70	150	1/20	4	1	2.5, 5.0
70	400	1/20	7	2	5.0
70	400	1/30	5.5	2	10.0, 15.0
70	400	1/60	4	2	15.0, 20.0

(Note that the technics are comparable to those used for chest roentgenography. The last column is discussed later in the paper.)

$x = 0$. This symmetry would not exist if the curve of x-ray intensity *vs.* time measured from the time $T/2$ were not symmetrical about $t = 0$.

The formulas listed above for E_α, E_β, and E_δ were calculated for the condition $\frac{x_2}{x_1} \geqslant 1$. If $\frac{x_2}{x_1} \leqslant 1$, a second set of formulas is required:

Constant Intensity Beam: $\frac{x_2}{x_1} \leqslant 1$

$$E_\alpha^* = \frac{a^2(I - I_0)}{2bfv(a + b)}\alpha^2 + I_0T \quad (0 \leqslant \alpha \leqslant x_2)$$

$$E_\beta^* = \frac{a(I - I_0)T}{bf}\beta + \frac{v(a + b)(I - I_0)T^2}{2bf} + I_0T \quad (0 \leqslant \beta \leqslant x_1 - x_2)$$

$$E_\delta^* = -\frac{a^2(I - I_0)}{2bfv(a + b)}\delta^2 + \frac{a(I - I_0)T}{bf}\delta - \frac{v(a + b)(I - I_0)T^2}{2bf} + IT \quad (0 \leqslant \delta \leqslant x_2).$$

These equations and those obtained by transformations like those described above for the E's when $\frac{x_2}{x_1} \geqslant 1$ show that the unsharpnesses on either side of the shadow of a moving absorbing strip are equal.

VI. TESTS OF UNSHARPNESS USING CHEST ROENTGENOGRAPHIC TECHNICS

Dr. G. S. Pesquera and his technical associate, Mr. E. C. Lasher, have constructed an instrument for making roentgenograms of pieces of lead which move parallel to the film with constant linear velocities. The lead strips are fastened to a disk of bakelite which is driven at constant angular velocity (one revolution per minute) by means of a motor, a speed-reducing gear train, and a belt; the edges of the six lead strips lie along a radius of the bakelite disk. The lead strips are so spaced, and the driving mechanism is so designed, that the linear velocities of the edges of the strips are 1.0, 2.5, 5.0, 10.0, 15.0, 20.0 millimeters per second. A photograph of this device is shown in a paper[7] recently published by Dr. Pesquera and Dr. Homer L. Sampson.

Dr. Pesquera has prepared several groups of films made with this device; he and several associates have visually compared the unsharpnesses of these films. He has sent the films and data to the author to be used in this paper. A short description of Dr. Pesquera's experiments and results follows.

The device described above was placed 20.3 cm. from the film. Five roentgenograms were made without intensifying screens by means of a four-valve apparatus with an impulse timer and a rotating anode tube using the technics shown in Table I.

For each exposure there were six unsharp shadow borders, for lead strips moving with linear velocities of 1.0, 2.5, 5.0, 10.0, 15.0, 20.0 mm. per sec. Thirty rectangular strips were cut from these films, each strip having a clear area (density about 0.14), an unsharp shadow of the moving edge of a lead strip, and a dark area (density about 1.0). These strips are like those shown in Figure 8 of the first paper. All strips which were the

[7] Pesquera, G. S., and Sampson, H. L.: The Evolution of Chest Roentgenographic Technic. Am. Jour. Roentgenol. and Rad. Ther., **40**, 405, September, 1938.

roentgenograms of the piece of lead moving at 1.0 mm. per sec. were collected in one group; a second group comprised films of the lead strip moving with a velocity of 2.5 mm. per sec.; there were six groups of five films each. Each of seven observers was asked to examine each group of films, and to choose two or three films which, in his opinion, had the least unsharpness. The data of these observers are summarized in Table II. Bold face numbers in this table are identification numbers of film strips; numbers in parentheses are calculated observed unsharpnesses in mm.; a bold face identification number, and the corresponding technic, may be correlated by referring to the data listed with the same bold face number in Table III. *The data of Table II were recorded before the observed unsharpnesses were calculated.*

TABLE II

Object Velocity (cm. per sec.)	Film Strip Identification Number and Calculated Observed Unsharpness (mm.)	Votes of Seven Observers		
		"most sharp of group"	"second most sharp of group"	"third most sharp of group"
1	**5** (0.13)	7	0	0
	12 (0.17)	0	5	0
	25 (0.18)	0	1	4
2.5	**11** (0.18)	7	0	0
	30 (0.19)	0	7	0
	4 (0.24)	0	0	3
	22 (0.23)	0	0	1
5.0	**8** (0.27)	4	3	0
	29 (0.25)	3	4	0
10.0	**23** (0.34)	5	2	0
	7 (0.50)	1	3	2
	26 (0.46)	0	2	1
	18 (0.34)	1	0	0
15.0	**20** (0.48)	4	3	0
	16 (0.36)	3	4	0
20.0	**17** (0.40)	7	0	0
	19 (0.63)	0	6	0

The data show that the seven observers' choices of the two or three most sharp roentgenograms of each group are in accord with determinations based upon calculations of the observed unsharpnesses, with two exceptions—film **18** and film **16**. The calculated observed unsharpnesses of these films are less than those of films chosen by the observers as more sharp. Two observers examined the two groups (v = 10.0 cm./sec. and v = 15.0 cm./sec.) again; the borders of films **18** and **16** appeared less sharp than other films of the same groups. Films **18** and **16** were examined by means of a low power (×2.5) microscope. The unsharp border appeared to comprise two narrow bands of high density perpendicular to the direction of motion of the lead strip; such striations were not perceptible in films **22, 4, 5, 24, 23.** It was concluded, therefore, that the two striations of films **18** and **16** were produced by the two pulsations of x-ray intensity of the 1/60 second exposure used for these films. *The observers' estimates of the unsharpnesses of films **16** and **18**, relative to the estimated unsharpnesses of other films for velocities of 10 cm. per sec. and 15 cm. per sec., are not in accord with the calculated unsharpnesses for films **16** and **18** because the intensity of the x-ray beam is not constant; the calculations are based upon the assumption of constant intensity.* The striations within the unsharp shadow border augment the appearance of unsharpness for exposures of 1/60 second; the effect is apparently negligible for exposures of 1/30 second or more for objects moving with velocities comparable to the velocities of objects within the chest. The apparent unsharpness of the shadow of a moving object may be greater for an exposure of 1/60 second than for some longer exposure. Therefore, exposures less than 1/30 second should not be used for objects having velocities from 5 cm./sec. to 15 cm./sec. if full wave rectified pulsating current flows in the roentgen-ray tube.

The values of the observed unsharpness listed in Tables II and III were calculated by means of four formulas:

$$U_0 = x_2 + x_1 \qquad 9 \leqslant \frac{x_1}{x_2} \leqslant \infty$$

$$U_0 = \frac{7}{16}\left(1 + \frac{1}{7}\frac{x_1}{x_2}\right)(x_2 + x_1)$$

$$1 \leqslant \frac{x_1}{x_2} \leqslant 9$$

$$U_0 = \frac{7}{16}\left(1 + \frac{1}{7}\frac{x_2}{x_1}\right)(x_2 + x_1) \qquad 1 \leqslant \frac{x_2}{x_1} \leqslant 9$$

$$U_0 = x_2 + x_1 \qquad 9 \leqslant \frac{x_2}{x_1} \leqslant 16.4$$

(16.4 is the highest value of x_2/x_1 in these data.)

The formulas differ from the formulas given in the original paper, which were:

$$U_0 = \left(1 - \frac{1}{2}\frac{x_2}{x_1}\right)(x_2 + x_1) \qquad 0 \leqslant \frac{x_2}{x_1} \leqslant 1$$

$$U_0 = \frac{1}{4}\left(1 + \frac{x_2}{x_1}\right)(x_2 + x_1) \qquad 1 \leqslant \frac{x_2}{x_1} \leqslant 3.$$

All of these formulas were chosen somewhat arbitrarily to correlate visual estimates of the relative unsharpnesses of test films; the formulas of the original paper[1] correlated the data described in that paper; the more extensive data now available are better correlated by the four formulas listed above and used to calculate the unsharpnesses of Table II and Table III. A brief description of the reasons for using these formulas follows:

The experimental films of Dr. Pesquera and Mr. Lasher have total unsharpnesses from 0.15 mm. to 2.41 mm.; the values of x_2/x_1 for the thirty exposures extend through the range $0.05 \leqslant \frac{x_2}{x_1} \leqslant 16.4$. Several films were held on the surface of a film illuminator, with a transparent scale (with divisions separated by 1 mm.), and examined by means of a low power (×2.5) microscope. The unsharpnesses (in millimeters) estimated in this manner were compared to the corresponding "observed unsharpnesses" calculated by means of the formulas of the original paper[1]:

$$U_0 = \left(1 - \frac{1}{2}\frac{x_2}{x_1}\right)(x_2 + x_1) \qquad 0 \leqslant \frac{x_2}{x_1} \leqslant 1$$

$$U_0 = \frac{1}{4}\left(1 + \frac{x_2}{x_1}\right)(x_2 + x_1) \qquad 1 \leqslant \frac{x_2}{x_1} \leqslant 3.$$

This comparison of visual estimates and calculated values shows that:

1. The calculated values and the visually estimated values of the "observed unsharpness" are approximately equal for the range $0.8 \leqslant \frac{x_2}{x_1} \leqslant 1.2$.
2. Visually estimated unsharpness is less than the unsharpness calculated by means of the two formulas above, when $\frac{x_2}{x_1} > 1.2$ and when $\frac{x_2}{x_1} < 0.8$.

Therefore, it was concluded that formulas might be chosen to correlate the experimental data more accurately than they are correlated by the formulas above. Examination of the films for which $\frac{x_2}{x_1} > 3$ shows that visually estimated unsharpness does not equal the calculated "total unsharpness" $(x_2 + x_1)$ until $\frac{x_2}{x_1} = 9$. This fact is clearly perceptible in Dr. Pesquera's films for which $(x_2 + x_1) < 2.5$ mm.; it is not demonstrated by the original test films[1] for which $(x_2 + x_1) = 10$ mm. Furthermore, the exposure formulas given in the first paper and in Section V show that the exposure is the same for any x whether $\frac{x_2}{x_1} = A (1 \leqslant A \leqslant 9)$ or $\frac{x_2}{x_1} = \frac{1}{A}$. The new formulas are chosen to take account of this fact. According to these new observations, therefore, the observed unsharpness may be more accurately calculated by means of the four formulas listed at the beginning of the last paragraph than by means of the formulas listed at the beginning of this paragraph.

VII. UNSHARPNESS OF THE SHADOW OF A MOVING OBJECT WITH X-RAY BEAM INTENSITY WHICH VARIES EXPONENTIALLY WITH TIME

The material described in the first paper[1] and in the first two sections of this paper is based upon the assumption that the intensity of the x-ray beam is constant during the exposure. It was shown in Section VI that the use of 1/60 second exposures with full wave rectified x-ray tube current and voltage produces results which are not in accord with the analysis because the x-ray intensity varies during the exposure. However, the data show that, for object velocities of the order of 5.0 cm./sec. to 15 cm./sec., the variations of intensity during exposures of 1/30 second or more do not produce a perceptible deviation from the analytic results. Finally, it was shown in Section V that intensity *vs.* time curves which are symmetrical about time $T/2$ produce equivalent unsharp shadow borders for both sides of a moving absorbing strip.

An example of an intensity *vs.* time curve which is not symmetrical about time $T/2$ is the output of a condenser-discharge roentgenographic apparatus. Such equipment has been used with discharge control apparatus of several types. Apparatus having mechanical switches for connecting the charged condenser to the x-ray tube have x-ray tube voltage and current wave forms which vary approximately exponentially with time. The wave forms

TABLE III

Focal spot-film distance (ft.)	4	5.5	7	4	5.5
Length of side of effective focal spot (mm.)	2	2	2	1	1
Exposure time (sec.)	1/60	1/30	1/20	1/20	1/10
Film Number	**14**	**21**	**25**	**12**	**5**
Linear velocity (mm./sec.)	1	1	1	1	1
x_2/x_1	0.05	0.14	0.26	0.30	0.82
U_0 (mm.)	0.42	0.27	0.18	0.17	0.13
Estimate of unsharpness (1 judged most sharp) S. R. W.	5	4	3	2	1
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	5	4	3	2	1
Film Number	**15**	**22**	**30**	**11**	**4**
Linear velocity (mm./sec.)	2.5	2.5	2.5	2.5	2.5
x_2/x_1	0.13	0.34	0.66	0.75	2.06
U_0 (mm.)	0.43	0.23	0.19	0.18	0.24
Estimate of unsharpness (1 judged most sharp) S. R. W.	5	4	2	1	3
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	5	4	2	1	3
Film Number	**13**	**24**	**29**	**8**	**6**
Linear velocity (mm./sec.)	5	5	5	5	5
x_2/x_1	0.25	0.68	1.31	1.50	4.12
U_0 (mm.)	0.34	0.25	0.25	0.27	0.49
Estimate of unsharpness (1 judged most sharp) S. R. W.	4	3	1	2	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	4	3	2	1	5
Film Number	**18**	**23**	**26**	**7**	**3**
Linear velocity (mm./sec.)	10	10	10	10	10
x_2/x_1	0.50	1.36	2.60	3.00	8.20
U_0 (mm.)	0.34	0.34	0.46	0.50	1.21
Estimate of unsharpness (1 judged most sharp) S. R. W.	4	1	2	3	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	4	1	2	3	5
Film Number	**16**	**20**	**28**	**9**	**1**
Linear velocity (mm./sec.)	15	15	15	15	15
x_2/x_1	0.75	2.04	4.00	4.50	12.4
U_0 (mm.)	0.36	0.48	0.71	0.85	1.85
Estimate of unsharpness (1 judged most sharp) S. R. W.	2	1	3	4	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	2	1	3	4	5
Film Number	**17**	**19**	**27**	**10**	**2**
Linear velocity (mm./sec.)	20	20	20	20	20
x_2/x_1	1.00	2.73	5.20	6.00	16.4
U_0 (mm.)	0.40	0.63	1.00	1.14	2.41
Estimate of unsharpness (1 judged most sharp) S. R. W.	1	2	3	4	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	1	2	4	3	5

for apparatus with electronic switches are also approximately exponential. In some condenser-discharge equipments the condenser is always connected to the x-ray tube and the exposure is produced by increasing quickly the temperature of the x-ray tube cathode; in others an inductance (a choke coil) is connected in series with the tube and condenser; the wave forms of voltage and current in these cases are not exponential, and they are different from each other. The discussion which follows is concerned solely with condenser-discharge apparatus in which the x-ray tube voltage and the x-ray tube current vary exponentially with time.

The exposure ratings of x-ray tubes used with condenser-discharge equipment have not been satisfactorily standardized. Some experiments suggest that tubes will safely withstand higher exposures (greater watt-seconds) when the energy is supplied by a charged condenser than they will withstand when the energy is supplied directly by means of a transformer and rectifier. Tube ratings often are based on the assumption that the energy of the exposure determines the rating, regardless of the kind of power-supply system. Because of this lack of agreement, and for other reasons stated below, no attempt is made in the following material to specify optimum exposure technics for condenser-discharge equipment.

It is the purpose of this Section (VII) to show that the methods described in Sections I to VI may be applied to exposure technics for which the x-ray intensity varies exponentially with time. It may be inferred from the results of this Section that the general methods of calculating unsharpness may be applied to any form of intensity *vs.* time variation; such calculations may be complicated from the algebraic point of view but they are not likely to be perplexing from the theoretical point of view.

The equations for calculating the ex-

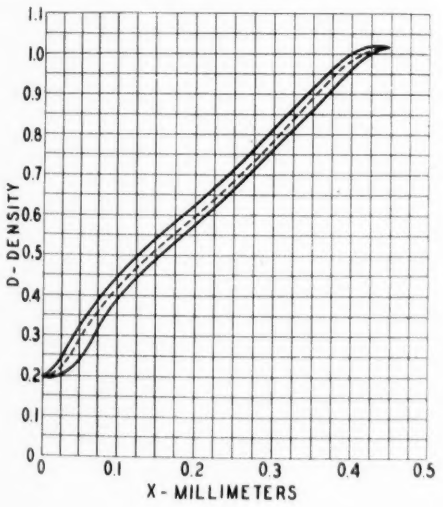

Fig. 11.

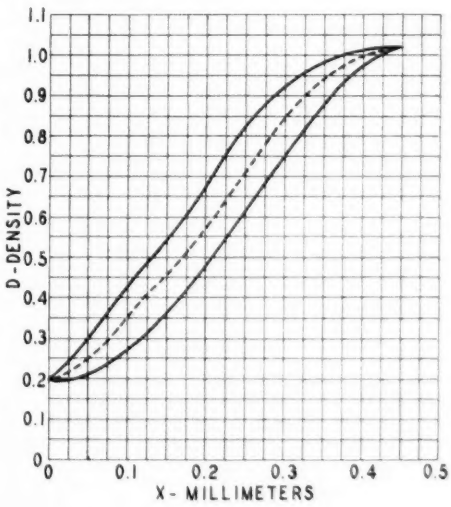

Fig. 12.

Fig. 11. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.2$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.2$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 0.2$; curve for either leading edge or trailing edge of absorbing strip.

Fig. 12. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.8$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.8$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 0.8$; curve for either leading edge or trailing edge of absorbing strip.

posure at any point from x_0 to x_3 in Figure 1 are derived as follows: The procedure is similar to that described in Section I of the first paper.[1] The x-ray intensity at the surface of the film is assumed to vary exponentially with time; the initial intensity at a point on the film "covered" by the object is I_0'; the initial intensity at a point on the film not "covered" by the object is I'; the roentgenographic exposure is assumed to end at the instant when the intensity has decreased to one-tenth of the initial intensity. Therefore:

$$i_0' = I_0' \epsilon^{-\frac{2.3t}{T}} \qquad i' = I' \epsilon^{-\frac{2.3t}{T}}$$

$i_0' = I_0'$ when $t = 0$

$i' = I'$ when $t = 0$

$i_0' = 0.1 I_0'$ when $t = T$

$i' = 0.1 I'$ when $t = T$.

The exposure at x_0 (Fig. 1) is:

$$E_{x_0}' = I_0' \int_0^T \epsilon^{-\frac{2.3t}{T}}\, dt = \frac{I_0' T}{2.3}(1 - 0.1) = \frac{I_0' T}{2.55}.$$

The exposure at x- x_3 is:

$$E_{x_3}' = I' \int_0^T \epsilon^{-\frac{2.3t}{T}}\, dt = \frac{I' T}{2.55}.$$

From this point the analysis proceeds in the same manner as that described in Section I. This analysis is straightforward, though somewhat tedious; the detailed description is therefore omitted. The expressions for calculating the exposure at any point from x_0 to x_3, for $\frac{x_2}{x_1} \geqslant 1$ are:

$$E_\alpha' = \frac{v(a+b)(I' - I_0')}{bfa_0^2}(\epsilon^{a_0' x} - 1) - \frac{a(I' - I_0')}{bfa_0}\alpha + \frac{I_0'}{a_0}(\epsilon^{a_0 T} - 1) \qquad (0 \leqslant \alpha \leqslant x_1)$$

where $a_0 \equiv \frac{-2.3}{T}$ and $t_\alpha = \frac{a\alpha}{v(a+b)}$

$$E_\beta' = \frac{v(a+b)(I' - I_0')}{bfa_0^2}(\epsilon^{a_0' \beta_2} - \epsilon^{a_0' \beta_1}) - \frac{I'}{a_0} + \frac{I_0'}{a_0}\epsilon^{a_0 T} \qquad (0 \leqslant \beta \leqslant x_2 - x_1)$$

where $a_0 \equiv -\frac{2.3}{T}$ and $t_{\beta_2} = \frac{a(x_1 + \beta)}{v(a+b)}$

and $t_{\beta_1} = \frac{a\beta}{v(a+b)}$

$$E_\delta' = \frac{v(a+b)(I' - I_0')}{bfa_0^2}(\epsilon^{a_0 T} - \epsilon^{a_0' \delta}) + \frac{a(I' - I_0')}{bfa_0}\epsilon^{a_0 T}\delta - \frac{I'}{a_0} + \frac{I_0'}{a_0}\epsilon^{a_0 T} \qquad (0 \leqslant \delta \leqslant x_1)$$

where $a_0 \equiv -\frac{2.3}{T}$ and $t_\delta = T - \frac{bf}{v(a+b)} + \frac{a\delta}{v(a+b)}$.

Expressions have been calculated for E_α', E_β', and E'_δ for cases in which $\frac{x_2}{x_1} \leqslant 1$. These equations are not listed here because it is the purpose of this Section to show the form of the results rather than the details of the results.

It was pointed out in Section V that an intensity *vs.* time curve which is not symmetrical about $T/2$ might be expected to produce dissimilar unsharpnesses of the shadow borders of a moving absorbing strip. The formulas for the exposures of points on the film for the case of the object placed *above* t_0 (Fig. 1) at the beginning of the exposure and moved upward during the exposure may be obtained by transforming the above equations for E_α', E_β', and E_δ' in a manner similar to that described in Section V. The results are not reproduced here, but their forms are illustrated in Figures 11, 12, 13, and 14 which are described below.

It is important to know what form of density *vs.* x curve is obtained from the formulas E_α', E_β', and E_δ', and film data like those described in Section II. Calculations were made for $(x_1 + x_2) = 0.45$ cm., using several values of x_2/x_1. Numerical values of I' and I_0' were so chosen that $E_{x_0}' = 0.15$ and $E_{x_3}' = 3$; data for

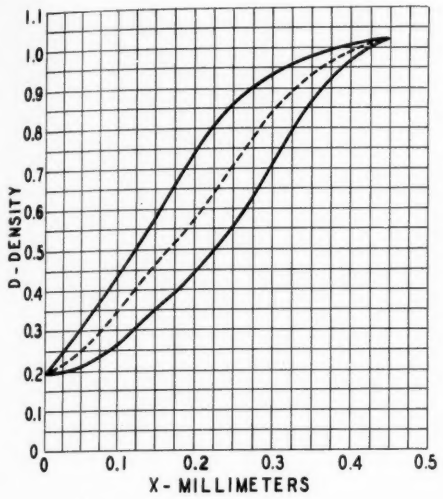

Fig. 13.

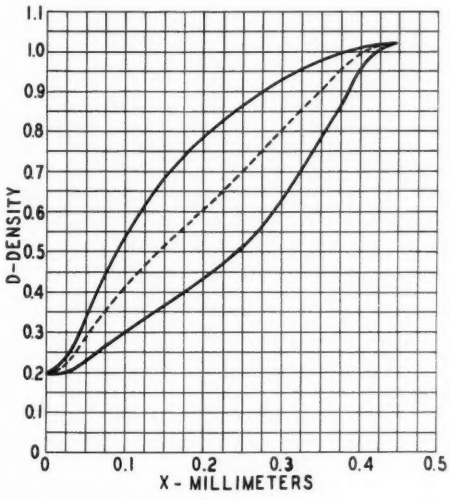

Fig. 14.

Fig. 13. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 1.5$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 1.5$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 1.5$; curve for either leading edge or trailing edge of absorbing strip.

Fig. 14. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 4.0$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 4.0$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 4.0$; curve for either leading edge or trailing edge of absorbing strip.

the film described in Section II indicate that these exposure values correspond to $D_{x_0} = 0.2$ and $D_{x_3} = 1.02$. Figures 11, 12, 13, 14 show graphically the results of these calculations for values of x_2/x_1 as follows: Figure 11, $x_2/x_1 = 0.2$; Figure 12, $x_2/x_1 = 0.8$; Figure 13, $x_2/x_1 = 1.5$; Figure 14, $x_2/x_1 = 4.0$. The top curve in each figure represents the change of density with x from x_0 upward for the conditions shown in Figure 1; the bottom curve in each figure represents the change of density with x from x_3 downward for the case when the object is *above* t_0 when $t = 0$; the dotted curve (between the top and bottom curves) represents the change of density with x for either original position of the object when the x-ray beam intensity is constant during the exposure.

These data point to the following conclusions:

1. The unsharp shadow borders of objects for which $x_2/x_1 \ll 1$ are nearly alike for constant intensity exposures and for exposures the intensities of which vary exponentially with time.
2. When $x_2/x_1 > 1$ the shadow borders of constant intensity exposures differ greatly from the borders of exponentially varying exposures. Furthermore the shadow

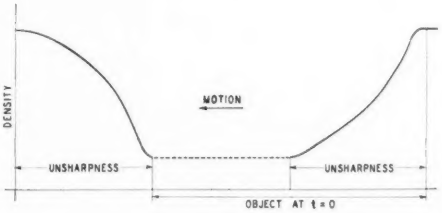

Fig. 15. Curve showing the estimated variation of density with distance along the shadow of a moving strip with condenser-discharge apparatus. Note that the shapes of the two curves of the unsharp shadow borders are quite similar to those shown in Figure 14. Figure 14 shows the calculated curves of the unsharpness for $x_2/x_1 = 4.0$; Figure 15 shows the visually estimated unsharpness curve for a roentgenogram made with condenser-discharge apparatus for $x_2/x_1 = 4.0$.

borders of opposite edges of an absorbing strip differ greatly if the intensity varies exponentially. Roentgenographic exposures of Pesquera's apparatus were made by Wilsey to test these conclusions.

The unsharp shadow borders of moving lead strips have the characteristics predicted by the analysis given above. If x_2/x_1 is less than about 0.5, the unsharpness appears to be a region of approximately uniform density gradient, from the point of minimum density to the point of maximum density; the unsharpnesses on either side of the shadow of the moving strip appear to be equivalent. If x_2/x_1 is greater than about 0.5, the shadows on either side of the moving strip differ greatly. For $x_2/x_1 = 4$ the shadow of the strip appears to have the form shown in Figure 15; this appearance corresponds to the analytic form of Figure 14.

SUMMARY

Additional experimental data and critical comments submitted to the author by other workers have been used to extend the scope of the analysis of the unsharpness of the shadow of a plane-absorbing object which moves with constant velocity parallel to the film during roentgenographic exposures. The following results are described briefly in terms of the analysis and experiments described in a paper[1] published in 1937:

1. The shadow of a moving absorbing strip is symmetrical about the center line of the shadow if the x-ray beam intensity is constant during exposure; in general, the shadow is symmetrical if the intensity *vs.* time curve is symmetrical about the time $T/2$.

2. Formulas are given for calculating the exposure (constant intensity) at any point in the area of unsharpness for $x_2/x_1 \leqslant 1$.

3. Tests by Pesquera suggest a modification of the formulas of U_0, presented in the first paper, to improve the accuracy of calculated observed unsharpnesses.

4. Pesquera submitted his test films to several experienced observers who were, however, unfamiliar with calculated unsharpnesses at the time they examined the films. Their observations confirm the analysis except for films made in 1/60 second at velocities greater than 5 cm./sec.; the intensity variations for such exposures preclude the use of the analytic formulas to predict the form of their unsharpnesses.

5. It is shown that a particular asymmetrical intensity *vs.* time curve that varies exponentially with time may be used as a basis for calculating the unsharpness for condenser-discharge roentgenographic apparatus. These calculations are found to be in accord with experimental data prepared by Wilsey, using condenser-discharge apparatus.

CASE REPORT

OSTEOGENIC SARCOMA OCCURRING IN FRAGILITAS OSSIUM[1]

A CASE REPORT

By F. C. JEWELL, M.D., *and* J. E. LOFSTROM, M.D., *Detroit, Michigan*

From the City of Detroit Receiving Hospital

The occurrence of osteogenic sarcoma and fragilitas ossium is not uncommon when alone, but a search through the literature failed to reveal a record of a case in which the two appeared together.

[1] Accepted for publication in May, 1939.

Lobstein, in 1833, first used the term "osteopsathyrosis" to denote idiopathic as well as other types of fragility of the bones. Looser later called it "osteogenesis imperfecta congenita" and "osteogenesis imperfecta tarda," the former being prenatal and affecting the bones of the skull and causing a large number of fractures of all the bones; and the latter being postnatal and affecting chiefly long bones. The pathologic picture was described as the same in both by Cinelli (1) and Ford (3).

The microscopic picture of fragilitas ossium shows a deficient functional activity of the osteoblasts. The preliminary processes of

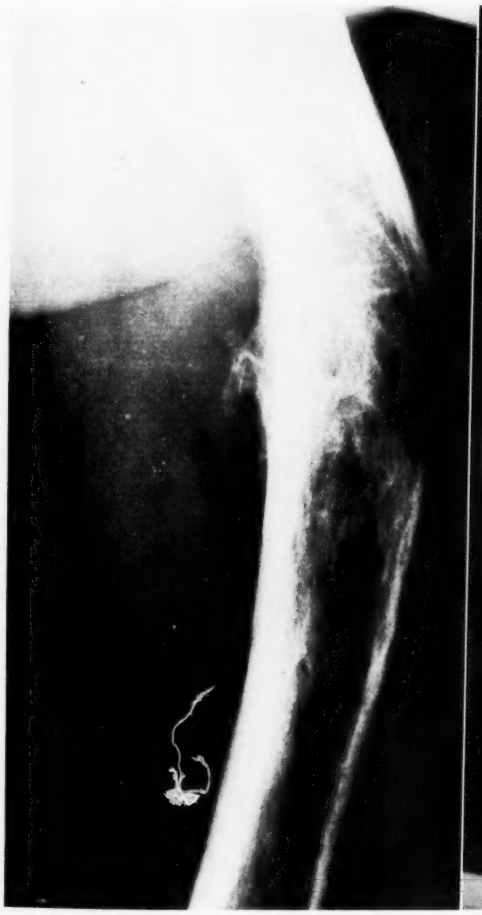

Fig. 1.

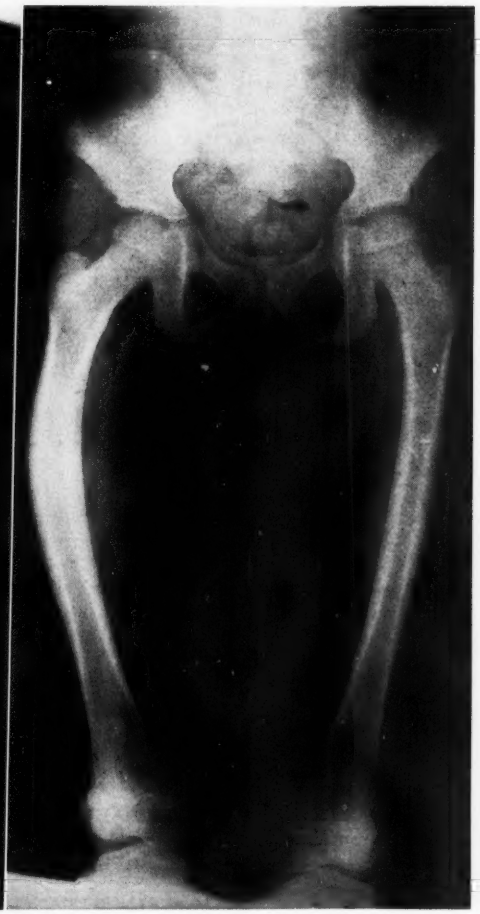

Fig. 2.

Fig. 1. Right femur showing old fracture and thinning of the cortex inferiorly.
Fig. 2. Femur of the patient's daughter, showing old fracture; increased caliber of medullary canals; deformity.

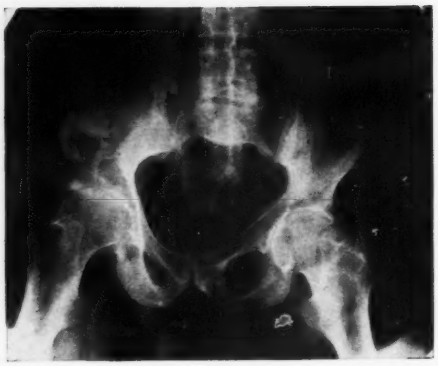

Fig. 3. Osteolytic process involving right ilium and upper half of acetabulum.

ossification are normal. There has been no general agreement as to any primary abnormality in the marrow or in the function of the osteoblasts. Metaplasia is frequent but probably compensatory, not primary (Bronson, 2). It is generally felt now, although we know nothing of the etiology, that osteogenesis imperfecta and fragilitas ossium are two separate pictures, the first being non-hereditary and the latter hereditary. There is no evidence to denote any change in the blood levels of phosphatase, chloride, phosphorus, or calcium in fragilitas ossium. The diagnosis of a case of fragilitas ossium showing an associated osteogenic sarcoma prompted us to make the following report.

Report of Case.—E. W., white male, 49 years of age, was admitted to the City of Detroit Receiving Hospital on Feb. 2, 1939, with the complaint of pain in the right hip. The patient stated that his present illness started in February, 1938, when he noted a dull, non-radiating pain localized in the region of the right hip. Symptoms were present on motion and from one to three hours after retiring. The condition had slowly progressed for the past year. Occasionally he was unable to work because of the condition of his hip. On Feb. 2, 1939, he fell on the ice, injuring his right hip. He was unable to rise and by virtue of his brittle bones he felt he had fractured his femur.

Past Illness.—At the ages of 7, 18, and 35 years the patient had fractured his right femur from minor trauma to the area (Fig. 1). At the age of 21, he had fractured his left femur. It is interesting to note that the patient's father had had his femora fractured several times and is now a cripple. A brother had had at least 12 fractures of the thigh and leg bones. One of the patient's daughters sustained two fractures of the right femur (Fig. 2). His grandmother suffered the same condition, and, in short, the brittle bones could be traced verbally for from 200 to 300 years in his family. The past history was otherwise irrelevant except that his teeth had become carious and removal had been necessary at the age of 18. He had always had blue sclera, and, from the age of 20, he had been slowly becoming deaf. There had been no familial history of carcinoma. His mother expired of pulmonary tuberculosis.

Physical Examination.—This revealed the patient to be well nourished and well developed. Skin, negative; head, negative; eyes, blue sclera; ears, otosclerotic deafness, both drums perforated; nose, negative; mouth, teeth removed; neck, negative; lymph glands, negative; heart and lungs, negative; vascular system, blood pressure, 130/85; abdomen, a hard, immovable mass in the right lower quadrant extending upward from the inguinal region a distance of 7 cm. The surface was smooth and attached to the underlying tissues. Bones, moderate tenderness of the right ilium 10 cm. from the symphysis. Some lateral bowing of the femora was noted.

Laboratory Examination.—Urine showed occasional white blood cells. Klein test was negative. Hemoglobin, 12.5 grams per cent; white blood cells, 8,500; red blood cells, 4.15 millions; filamentous neutrophils, 64 per cent; nonfilamentous neutrophils, 12 per cent; lymphocytes, 24 per cent. Blood phosphatase, 5.5 units; blood chloride, 462 mg./100 c.c.; serum phosphorus, 4.4 mg./100 c.c.; serum albumin, 3.5 per cent; serum globulin, 3.1 per cent; blood calcium, 9.4 mg.; sedimentation rate, 12 minutes.

Roentgenographic Findings.—The right femur (Fig. 1) reveals evidence of old healed fractures at the junction of the upper and middle thirds and extending to the region of the lesser trochanter. Union is complete with moderate lateral bowing. In addition, there is thinning of the femoral cortex especially on its lateral aspect throughout the entire length of the shaft. The medullary canal is widened. Similar findings were noted in the left femur. Roentgenograms of the skull, chest, thoraco-lumbar spine, and humerus showed no abnormalities.

The pelvis (Fig. 3) shows evidence of an area of osteolysis involving the right ilium and also the upper half of the acetabulum, with disruption of the articular surface. The area extends in an irregular manner superiorly to the vicinity of the anterior superior spine and also through the medial wall of the ilium. There are a few islets of bone remaining within the lytic area. There is no new bone production. There is a soft-tissue mass visible, extending laterally and also into the pelvic cavity. There is slight abnormality of the contour of

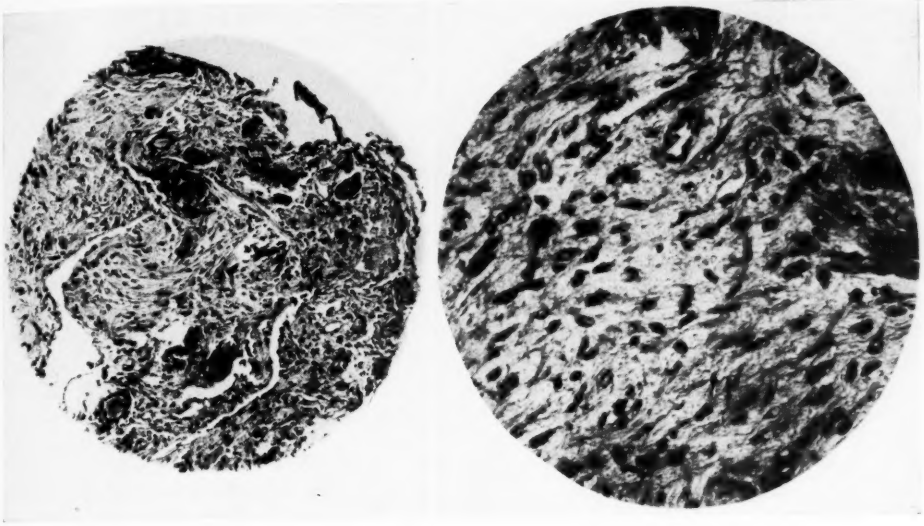

Fig. 4. Fig. 5.

Fig. 4. Biopsy of neoplastic areas composed of spindle cells containing osteoid deposits with calcification of atypical trabeculæ (low power).

Fig. 5. Spindle-shaped neoplastic cells differentiated into osteoblasts (high power).

the head of the femur which is due to faulty weight-bearing secondary to fractures of the femur. The osseous structure is otherwise normal.

Roentgen examination shows moderate lateral bowing in the middle and upper thirds of the right femur of the patient's daughter (Fig. 2). The cortex in this instance is not noticeably thinned although the medullary canal is of increased caliber. There is resultant shortening of the shaft and slight widening of the distal end of the femur at the epiphysis. In view of the familial history and the findings presented by the father, it was considered that these were of the nature of fragilitas ossium.

It was felt that the process in the right ilium was due to an area of primary malignancy. Sarcoma, lymphoblastoma, and Ewing's tumor were considered in the differential diagnosis. The condition of the femora with associated fractures and familial history is relatively typical of fragilitas ossium.

On Feb. 21, 1939, a biopsy was taken from the mass over the right inguinal region. The report given by Dr. O. A. Brines, pathologist, is as follows (Figs. 4 and 5): "The sections represent a neoplasm composed chiefly of spindle-shaped cells embedded in an abundant mucinous stroma. In some areas the spindle neoplastic cells differentiate into osteoblasts, and small deposits of osteoid material have been formed which in some instances assume a definite trabecular pattern. In some of these trabeculæ calcification has occurred." Following this it was the decision of the Tumor Clinic that this patient should have a course of deep x-ray therapy.

SUMMARY

1. A case of associated fragilitas ossium and osteogenic sarcoma is presented.

2. Although these two conditions occurred together, it is not felt that there is any direct relationship between them.

3. The familial history of the fragilitas ossium can be traced verbally for two or three hundred years.

4. A review of the literature failed to reveal any similar relationship in the pathologic conditions.

REFERENCES

(1) Cinelli, A. A.: Fragilitas Ossium with Otosclerosis and Blue Sclera: Report of a Case. Arch. Otolaryng., **25,** 309–311, March, 1937.

(2) Bronson, E.: Fragilitas Ossium and its Association with Blue Sclera and Otosclerosis. Edinburgh Med. Jour., **18,** 240–281, April, 1917.

(3) Ford, S.: Congenital Bone Dysplasia. Radiology, **30,** 117–121, January, 1939.

THE WASHINGTON SCENE

Although the so-called National Health Bill drafted by Senator Wagner last year failed to reach the Senate floor, reports from some sources are that it is currently being revised and will be reintroduced before the Seventy-sixth Congress adjourns. Meanwhile, various bills intended to accomplish certain aims of the original bill have been introduced in the national Congress.

The Wagner-George Bill, *S. 3230*, upon which hearings have already been held, called for appropriation of $10,000,000 the first year for construction of hospitals in needy areas, title to remain in the Federal Government. The Mead Bill, *S. 3246*, proposed to authorize a federal appropriation of $300,000,000 for loans to States for hospitals, water, sewers, stream pollution control, and related projects and facilities. Of the total, $100,000,000 was to be devoted to hospital projects. Senator Mead, in his bill, defines a hospital as any institution for treating disease, including any health, diagnostic, or treatment center, station, or clinic.

A bill introduced by Senator Taft, of Ohio, on April 18, embodies certain of the recommendations contained in a memorandum left by representatives of the American Medical Association following a conference in Washington on January 10. It would provide that the Federal Government contribute, for a period of five years and from annual appropriations of $10,000,000, from 40 to 90 per cent of the cost of hospitals constructed with the approval of a National Advisory Hospital Council appointed by the Surgeon-General with the approval of the Federal Security Agency. Title to the hospitals would rest in the local jurisdiction.

In all these bills, equipment to be installed in the hospital is contemplated as a part of the cost of construction, it seems. In the case of the Wagner-George Bill, at least, this would entail a certain amount of governmental supervision in the use of this equipment. The status of radiologists operating in such hospitals is a matter for conjecture.

A revised version of the Wagner-George Bill was reported out for the Senate Committee on Education and Labor, by Senator James E. Murray, of Montana, on April 30. The Committee frankly states in its report that the new bill is designed to accomplish part of the original Wagner Health Bill and that it is only the initial step in the Committee's projected program.

The revised bill would appropriate $10,000,000 the first fiscal year to be used for hospital construction and an equal amount for each of the five succeeding years to be used for grants-in-aid to States and subdivisions thereof for construction and maintenance of hospitals. It also authorizes the use of 2 per cent of the appropriations for the training of personnel necessary for such hospitals. Title can be acquired by the local County or State. The proposal expressly states that the hospitals are to be used for "diagnosis."

Kansas' Senator Arthur C. Capper is less modest in the proposals contained in his bill. Introduced, on March 25, it is the revised Epstein Bill and calls for $50,000,000 the first year and indeterminate amounts in succeeding years to induce States to establish systems of compulsory health insurance under the Social Security Act.

Senator Henry Cabot Lodge, of Massachusetts, introduced, on March 19, a bill to provide for federal health insurance program and the furnishing of "those medical services and facilities which have become standardized in their nature, but which because of high cost are not used in many cases in which their use would be unquestionably desirable."

In discussing his bill, Senator Lodge said: "I believe it is not disputed that countless instances occur every day in which x-ray examinations are desirable—nay, essential—but are not given because of the prohibitive cost. The suffering which could be prevented by prompt x-ray examination is indescribable. Needless to say, the prevention of disease automatically tends to reduce the cost of caring for the disease once it has been allowed to take hold. The use of respirators and x-rays in-

volves a technic which has become relatively standardized. In the case of the x-ray, it is not inconceivable that it would become a routine part of every physical examination were it not for the cost. In the case of persons of moderate means the cost prohibits its use. In the case of persons who can afford it, the use of x-ray is known to be so unusual that its prescription sometimes causes alarm."

A fellow Bostonian, Dr. Hugh Cabot, adds fuel to the flame by continuing his self-appointed rôle of propagandist for medical reform. He is the author of an article in the April issue of the *American Magazine*, in which he washes some of his own dirty linen and concludes that the best way to prohibit the repetitions of such ulterior practices, as he confesses, is to socialize medicine. He is apparently unaware of the national examining boards, like the American Board of Radiology, for he leaves the impression that any doctor may set himself up as a specialist without any special qualifications. He omits any mention of the fact that strenuous efforts are being made by organized medicine, without the need for governmental regimentation, to police its own members and raise the standards for specialty practice.

Says the *Journal of the Indiana Medical Association*, commenting editorially: "Dr. Cabot forgets that only a very small per cent of M.D.'s are surgeons. Most of the conditions for which patients seek relief are medical and functional—not surgical, and one of the first duties of a doctor is to relieve the patient of worry through personal contact. Dr. Cabot is not accustomed to this phase of medical practice—he is used to going to the operating room where the patient is completely ready for the surgery that he performs. He is not called upon to see the patient through the minor difficulties for which the physician collects one- and two-dollar fees. The big fees of the surgical specialist are best known to him and he has had little opportunity to know about the kind of medical practice that the average physician does—not the extremist or the ultra-conservative, but just the average physician."

MAC F. CAHAL *Executive Secretary*

RADIOLOGICAL SOCIETIES IN NORTH AMERICA

Editor's Note.—Will secretaries of societies please cooperate with the Editor by supplying him with information for this section? Please send such information to Leon J. Menville, M.D., 1201 Maison Blanche Bldg., New Orleans, La.

UNITED STATES

CALIFORNIA

California Medical Association, Section on Radiology.—Chairman, Karl M. Bonoff, M.D., 1930 Wilshire Blvd., Los Angeles; *Secretary,* Carl D. Benninghoven, M.D., 95 S. El Camino Real, San Mateo.

Los Angeles County Medical Association, Radiological Section.—President, M. L. Pindell, M.D.; *Vice-president,* Richard T. Taylor, M.D.; *Secretary,* Wilbur Bailey, M.D., 2007 Wilshire Blvd.; *Treasurer,* Henry Snure, M.D., 1414 South Hope Street; Kenneth Davis, M.D., *Member of Executive Committee.* Meets second Wednesday of each month at County Society Building.

Pacific Roentgen Club.—Chairman, Karl M. Bonoff, M.D., Los Angeles; *Members of Executive Committee,* I. S. Ingber, M.D., A. C. Siefert, M.D., D. R. MacColl, M.D.; *Secretary-Treasurer,* L. Henry Garland, M.D., 450 Sutter St., San Francisco. Executive Committee meets quarterly; Club meets annually during annual session of the California Medical Association.

San Francisco Radiological Society.—Secretary, Harold A. Hill, M.D., 450 Sutter Street. Meets monthly on third Thursday at 7:45 P.M., for the first six months at Toland Hall (Univ. of Calif. Med. School) and for the second six months at Lane Hall (Univ. of Stanford Med. School).

COLORADO

Denver Radiological Club.—President, N. B. Newcomer, M.D., 306 Republic Bldg.; *Vice-president,* Elizabeth Newcomer, M.D.; *Secretary,* Paul R. Weeks, M.D., 520 Republic Bldg.; *Treasurer,* L. G. Crosby, M.D., 366 Metropolitan Bldg. Meets third Friday of each month at homes of members.

The *Denver Radiological Club* will hold its Annual Midsummer Radiological Conference in the Rocky Mountains, with headquarters at the Hotel Shirley-Savoy, the dates of the meeting being August 8, 9, and 10.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Chairman, Samuel M. Atkins, M.D., 63 Central Ave., Waterbury; *Secretary-Treasurer,* Max Climan, M.D., 242 Trumbull St., Hartford. Meetings twice annually in May and September.

DELAWARE

Affiliated with Philadelphia Roentgen Ray Society.

FLORIDA

Florida Radiological Society.—President, H. B. McEuen, M.D., Jacksonville; *Vice-president,* Joseph H. Lucinian, M.D., Miami; *Secretary-Treasurer,* John N. Moore, M.D., 210 Professional Bldg., Ocala. Meetings held in November and at the annual meeting of the Medical Association of Florida in the spring.

GEORGIA

Georgia Radiological Society.—President, James J. Clark, M.D., Doctors Bldg., Atlanta; *Vice-president,* L. P. Holmes, M.D., University Hospital, Augusta; *Secretary-Treasurer,* Robert C. Pendergrass, M.D., Prather Clinic, Americus. Meetings twice annually, in November and at the annual meeting of the Medical Association of Georgia in the spring.

ILLINOIS

Chicago Roentgen Society.—President, Roe J. Maier, M.D.; *Vice-president,* Adolph Hartung, M.D.; *Secretary,* Chester J. Challenger, M.D., 3117 Logan Blvd. Meetings the second Thursday of each month from October to May, except December, at the Hotel Sherman.

Illinois Radiological Society.—President, Harry W. Ackeman, M.D., 321 W. State St., Rockford; *Vice-president,* D. R. Hanley, M.D., St. Mary's Hospital, Streator; *Secretary-Treasurer,* William DeHollander, M.D., St. John's Hospital, Springfield. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—Chairman, Warren W. Furey, M.D., 6844 Oglesby Ave., Chicago; *Secretary,* Harry W. Ackeman, M. D., 321 W. State St., Rockford.

INDIANA

The Indiana Roentgen Society.—President, Juan Rodriguez, M.D., 2902 Fairfield Ave., Fort Wayne; *President-elect,* H. H. Inlow, M.D., Shelbyville; *Vice-president,* Wemple Dodds, M.D., Crawfordsville; *Secretary-Treasurer,* Clifford C. Taylor, M.D., 23 E. Ohio St., Indianapolis. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Holds luncheon and business meeting during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—President, D. B. Harding, M.D., Lexington; *Vice-president,* I. T. Fugate, M.D., Louisville; *Secretary-Treasurer,*

Joseph C. Bell, M.D., 402 Heyburn Bldg., Louisville. Meeting annually in Louisville, third Sunday afternoon in April.

The Annual Meeting of the *Kentucky Radiological Society* was held at the Brown Hotel, Louisville, on Sunday, April 21, 1940. The meeting convened at 11:20 A.M. with D. B. Harding, M.D., *President*, presiding. Following the business session luncheon was served. After the luncheon Howard P. Doub, M.D., of Detroit, addressed the Society on the subject of "Childhood Tuberculosis." His presentation was illustrated by numerous lantern slides. Following Dr. Doub's address, cases illustrated by lantern slides were reported by members of the Society and were discussed by those present. The meeting adjourned at 5:30 P.M.

MAINE

See New England Roentgen Ray Society.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Chairman, Harold E. Wright, M.D., 101 W. Read St.; *Secretary*, Walter L. Kilby, M.D., 101 W. Read St. Meetings are held the third Tuesday of each month.

MASSACHUSETTS

See New England Roentgen Ray Society.

MICHIGAN

Detroit X-ray and Radium Society.—President, Sam W. Donaldson, M.D., 326 N. Ingalls St., Ann Arbor; *Vice-president*, Clarence Hufford, M.D., 421 Michigan St., Toledo, Ohio; *Secretary-Treasurer*, E. R. Witwer, M.D., Harper Hospital, Detroit. Meetings first Thursday of each month from October to May, inclusive, at Wayne County Medical Society club rooms, 4421 Woodward Ave.

Michigan Association of Roentgenologists.—President, J. H. Dempster, M.D., Detroit; *Vice-president*, L. E. Holly, M.D., Muskegon; *Secretary-Treasurer*, J. E. Lofstrom, M.D., 1536 David Whitney Bldg., Detroit. Meetings quarterly by announcement.

MINNESOTA

Minnesota Radiological Society.—President, Leo G. Rigler, M.D., University Hospital, Minneapolis; *Vice-president*, Harry M. Weber, M.D., Mayo Clinic, Rochester; *Secretary*, John P. Medelman, M.D., 572 Lowry Medical Arts Bldg., St. Paul. Meetings quarterly.

MISSOURI

The Kansas City Radiological Society.—President, L. G. Allen, M.D., 907 N. 7th St., Kansas City, Kansas; *Secretary*, Ira H. Lockwood, M.D., 306 E. 12th St., Kansas City, Mo. Meetings last Thursday of each month.

The St. Louis Society of Radiologists.—President, Oscar C. Zink, M.D., St. Luke's Hospital; *Secretary*, Wilbur K. Mueller, M.D., University Club Bldg. Meets on fourth Wednesday of October, January, March, and May, at a place designated by the president.

NEBRASKA

Nebraska Radiological Society.—President, Roy W. Fouts, M.D., 1007 Medical Arts Bldg., Omaha; *Secretary*, D. Arnold Dowell, M.D., 816 Medical Arts Bldg., Omaha. Meetings third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY

(Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island.) *President*, Langdon T. Thaxter, M.D., Maine General Hospital, Portland, Maine; *Secretary*, Aubrey O. Hampton, M.D., Massachusetts General Hospital, Boston. Meetings third Friday of each month from October to May, inclusive, usually at Boston Medical Library.

NEW HAMPSHIRE

See New England Roentgen Ray Society.

NEW JERSEY

Radiological Society of New Jersey.—President, P. S. Avery, M.D., Middlesex Hospital, New Brunswick; *Vice-president*, J. G. Boyes, M.D., 912 Prospect Ave., Plainfield; *Treasurer*, H. A. Vogel, M.D., 1060 E. Jersey St., Elizabeth; *Secretary*, W. James Marquis, M.D., 198 Clinton Ave., Newark; *Counsellor*, A. W. Pigott, M.D., Skillman. Meetings at Atlantic City at time of State Medical Society, and Midwinter in Newark as called by president.

NEW YORK

Associated Radiologists of New York, Inc.—President, I. J. Landsman, M.D., 910 Grand Concourse, New York City; *President-elect*, D. E. Ehrlich, M.D., 35 West 92nd St., New York City; *Vice-president*, Frederic E. Elliott, M.D., 122 76th St., Brooklyn; *Treasurer*, Solomon Fineman, M.D., 133 East 58th St., New York City; *Secretary*, William J. Francis, M.D., 210 Fifth Ave., New York City. Regular meetings the first Monday evening of the month in March, May, October, and December.

Brooklyn Roentgen Ray Society.—President, A. L. L. Bell, M.D., Long Island College Hospital, Henry, Pacific, and Amity Sts.; *Secretary-Treasurer*, L. J. Taormina, M.D., 1093 Gates Ave. Meetings first Tuesday in each month at place designated by president.

Buffalo Radiological Society.—President, Chester D. Moses, M.D., 333 Linwood Ave.; *Vice-president*, Edward C. Koenig, M.D., 100 High St.; *Secretary-Treasurer*, Joseph S. Gian-Franceschi, M.D., 610 Niagara St. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Ray Society.—President, Jesse Randolph Pawling, M.D., 305 Clinton St., Watertown; *Vice-president*, Albert Lenz, M.D., 613

State St., Schenectady; *Secretary-Treasurer*, Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse. Meetings are held in January, May, and October, as called by Executive Committee.

Long Island Radiological Society.—President, Samuel G. Schenck, M.D., Brooklyn; *Vice-president*, G. Henry Koiransky, M.D., Long Island City; *Secretary*, Marcus Wiener, M.D., 1430 48th St., Brooklyn; *Treasurer*, Louis Goldfarb, M.D., 608 Ocean Ave., Brooklyn. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—President, Harry M. Imboden, M.D., 30 W. 59th St., New York City; *Vice-president*, Henry K. Taylor, M.D., 667 Madison Ave., New York City; *Secretary*, Roy D. Duckworth, M.D., 170 Maple Ave., White Plains, N. Y.; *Treasurer*, Eric J. Ryan, M.D., St. Luke's Hospital, New York City.

Rochester Roentgen-ray Society.—Chairman, George H. S. Ramsey, M.D., 277 Alexander St.; *Secretary*, S. C. Davidson, M.D., 277 Alexander St. Meetings at convenience of committee.

NORTH CAROLINA

Radiological Society of North Carolina.—President, Robert P. Noble, M.D., 127 W. Hargett St., Raleigh; *Vice-president*, A. L. Daughtridge, M.D., 144 Coast Line St., Rocky Mount; *Secretary-Treasurer*, Major I. Fleming, M.D., 404 Falls Road, Rocky Mount. Meetings with State meeting in May, and meeting in October.

OHIO

Cleveland Radiological Society.—President, L. A. Pomeroy, M.D., Hanna Bldg., Cleveland; *Vice-president*, P. C. Langan, M.D., 215 Wellsley Ave., Akron; *Secretary-Treasurer*, H. A. Mahrer, M.D., 10515 Carnegie Ave., Cleveland. Meetings at 6:30 P.M. at the Mid-day Club, in the Union Commerce Bldg., on fourth Monday of each month from October to April, inclusive.

A notice of the joint meeting with the Pittsburgh Roentgen Society will be found under the latter Society, on this page.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*President*, Archie Fine, M.D., 707 Race St., Cincinnati; *Secretary-Treasurer*, Justin E. McCarthy, M.D., 707 Race St., Cincinnati, Ohio. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—President, Louis A. Milkman, M.D., Medical Arts Bldg., Scranton; *First Vice-president*, James E. Ginter, M.D., Dubois; *Second Vice-president*, Alexander Stewart, M.D., Shippensburg; *Secretary-Treasurer*, L. E. Wurster, M.D., 416 Pine St., Williamsport; *President-elect*, Harvey N. Mawhinney, M.D., 6546 Darlington Road, Pittsburgh; *Editor*, William E. Reiley, M.D., Clearfield; *Assistant Editor*, Sydney J. Hawley, M.D., Danville.

The Philadelphia Roentgen Ray Society.—President, Joseph E. Roberts, Jr., M.D., 403 Cooper St., Camden, N. J.; *Vice-president*, Jacob H. Vastine, M.D., Medical Arts Bldg., Philadelphia; *Secretary*, Barton R. Young, M.D., Temple University Hospital, Philadelphia; *Treasurer*, Fay K. Alexander, M.D., Chestnut Hill Hospital, Philadelphia. Meetings held first Thursday of each month at 8:15 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22nd St., Philadelphia.

The Pittsburgh Roentgen Society.—President, Zoe A. Johnston, M.D., 601 Jenkins Arcade; *Vice-president*, Prentiss A. Brown, M.D., and *Secretary-Treasurer*, Harold W. Jacox, M.D., 4800 Friendship Ave. Meetings held second Wednesday of each month at 4:30 P.M., from October to June at various hospitals designated by program committee.

On Monday, April 29, the members of the Cleveland Radiological Society were the guests of the *Pittsburgh Roentgen Society* at a meeting and dinner in the Hotel William Penn in Pittsburgh. Fifty-six members and guests attended and an excellent scientific program was presented.

Dr. Lloyd Craver, of the Memorial Hospital, New York City, spoke on the subject of Bronchiogenic Carcinoma. Dr. Charles Higley and Dr. Harry Hauser, of the Cleveland City Hospital, spoke on the Diagnosis and Treatment of Intrathoracic Hodgkin's Disease, respectively. Dr. Paul Bovard, of Tarentum, Pa., presented Diagnostic Problems Associated with Silicosis.

It is hoped that a combined meeting of the two societies will become an annual affair.

RHODE ISLAND

See New England Roentgen Ray Society.

SOUTH CAROLINA

South Carolina X-ray Society.—President, T. A. Pitts, M.D., Columbia; *Secretary-Treasurer*, Malcolm Mosteller, M.D., Columbia Hospital, Columbia. Meetings in Charleston on first Thursday in November, also at time and place of South Carolina State Medical Association.

SOUTH DAKOTA

Meets with Minnesota Radiological Society.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—President, Steve W. Coley, M.D., Methodist Hospital, Memphis; *Vice-president*, Eugene Abercrombie, M.D., 305 Medical

Arts Bldg., Knoxville; *Secretary-Treasurer*, Franklin B. Bogart, M.D., 311 Medical Bldg., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Texas Radiological Society.—President, C. F. Crain, M.D., Corpus Christi; *President-elect*, M. H. Glover, M.D., Wichita Falls; *First Vice-president*, G. D. Carlson, M.D., Dallas; *Second Vice-president*, P. E. Wigby, M.D., Dallas; *Secretary-Treasurer*, L. W. Baird, M.D., Scott and White Hospital, Temple. Meets annually. The next annual meeting is to be Jan. 18, 1941, in Sherman.

VERMONT

See New England Roentgen Ray Society.

VIRGINIA

Radiological Society of Virginia.—President, Fred M. Hodges, M.D., 100 W. Franklin St., Richmond; *Vice-president*, L. F. Magruder, M.D., Raleigh and College Aves., Norfolk; *Secretary*, V. W. Archer, M.D., University of Virginia Hospital, Charlottesville.

WASHINGTON

Washington State Radiological Society.—President, H. E. Nichols, M.D., Stimson Bldg., Seattle; *Vice-president*, George Cornett, M.D., Yakima; *Secretary-Treasurer*, Kenneth J. Holtz, M.D., American Bank Bldg., Seattle. Meetings fourth Monday of each month at College Club, Seattle.

WISCONSIN

Milwaukee Roentgen Ray Society.—President, H. W. Hefke, M.D.; *Vice-president*, Frederick C. Christensen, M.D.; *Secretary-Treasurer*, Irving I. Cowan, M.D., Mount Sinai Hospital, Milwaukee. Meets monthly on first Friday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, Russel F. Wilson, M.D., Beloit Municipal Hospital, Beloit. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society, in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 1300 University Ave., Madison, Wis. Meets every Thursday from 4 to 5 P.M., Room 301, Service Memorial Institute.

CANADA

Section on Radiology, Canadian Medical Association.—Chairman, Gordon Richards, M.D., Medical Arts Bldg., Toronto; *Secretary*, W. J. Cryderman, M.D., Medical Arts Bldg., Toronto.

Section on Radiology, Ontario Medical Association.—Chairman, E. H. Shannon, M.D., St. Michael's Hospital, Toronto; *Secretary*, W. J. Cryderman, M.D., 474 Glenlake Avenue, Toronto.

Canadian Association of Radiologists.—President, J. E. Gendreau, M.D., Montreal; *Vice-president*, W. H. McGuffin, M.D., Calgary; *Honorary Secretary-Treasurer*, W. L. Ritchie, M.D., Montreal; *Chairman of Interrelations Committee*, G. E. Richards, M.D., Toronto.

EDITORIAL

LEON J. MENVILLE, M.D., *Editor* HOWARD P. DOUB, M.D., *Associate Editor*

THE RADIOLOGIST'S PLACE IN THE DIAGNOSIS OF LUNG PATHOLOGY

A few years ago, when the radiologist was requested to make an x-ray examination of the chest and express an opinion, too often his findings were either accepted by the surgeon, internist, or general practitioner, as the final word and allowed to override conflicting clinical evidence, or they were minimized and the clinical evidence overemphasized. Twenty-five years ago, there were many good clinicians who sincerely believed that a careful physical examination by a competent chest man would afford an accurate diagnosis in most cases of early tuberculosis. They certainly believed that physical signs were far more important than the x-ray findings. As the fundamental knowledge on which an accurate interpretation of chest pathology as revealed in the roentgenogram became more widely known, the well trained clinician quickly recognized the important place which an x-ray examination of the chest occupied. Conversely, the good radiologist soon appreciated that, even if a great deal of early pathology in the lungs, which was overlooked by the physical examination, was detected by the roentgen examination, the x-ray examination in no way replaced the physical examination, and clinical history but only supplemented them.

As the years have passed, the proper place of the x-ray in the examination of the chest has been recognized by the great majority of both clinicians and radiologists. Unfortunately, there are still some clinicians and some men who are doing radiology, who do not appreciate what the proper place of the x-ray examination is in the determination and evaluation of chest pathology. About ten years ago, a patient was referred to the writer for an x-ray examination of the chest by a general practitioner. The findings were such as were at that time generally ascribed to healed miliary tuberculosis, although now we recognize the fact that they are likely produced by other processes. In the description of the film, the statement was clearly made that there was no soft infiltration, which was suggestive of active tuberculosis. In the space at the bottom of the report sheet reserved for the roentgen diagnosis, this statement was made, "healed miliary tuberculosis." Unfortunately, only the written report was rendered, and the case was not discussed with the clinician. A few weeks later, it was learned that the doctor had hurriedly glanced at the diagnosis and, overlooking the word "healed," had sent the patient to a tuberculosis sanatorium with a diagnosis of tuberculosis based on the x-ray findings.

This incident not only illustrates the fact that clinical diagnoses should be arrived at after consultation between the clinician and the radiologist, but that the same kind of report is not suitable when rendered to different types of referring physicians. In a large closed hospital or clinic in which there exist close understanding and co-operation between the different departments, a detailed description of the roentgen findings may not be necessary in every case, and only the doubtful cases need be carefully discussed in conference. In other institutions, where the size of the staff permits, it might well be argued that an accurate description should be rendered by the radiologist and that only in conference should any diagnosis be made. In the average community the problem of the radiologist may be quite different. He will undoubtedly have many referring physicians with whom he has a perfect understanding who will evaluate the x-ray report at its true worth and who will gladly consult with the radiologist in all doubtful cases. In spite of all the educational work which the competent radiologist can diplomatically do, there will be a considerable number of referring physicians who, because of indifference or haste, will depend on the radiologist's report almost exclusively to arrive at a clinical diagnosis. There will also be a few men who will fail to realize the necessity

for an x-ray examination and will not have it done unless the patient insists. Such a doctor will be apt to minimize the importance of the x-ray findings. Fortunately, such doctors are not so frequently encountered as they were a few years ago.

Another thing that must be taken into account in evaluating the proper place of the roentgen examination in the diagnosis of chest pathology is the fact that with the great increase in the number of cases coming for roentgen examination, and the widespread propaganda for early diagnosis, many early lesions are being encountered that were formerly not seen. The experienced radiologist encounters an increasing number of cases in which it is not only necessary to correlate the roentgen and clinical findings, but in which a diagnosis is possible only after repeated roentgen examinations at fairly frequent intervals and careful clinical checks.

With the recent widespread distribution of x-ray equipment and the interpretation of chest films by the comparatively inexperienced, shadows on the film that are of no particular significance are frequently interpreted as indicating the presence of grave pathology. This mistake occurs more commonly than for a serious lesion to be overlooked.

The good radiologist experiences no chagrin in frankly stating in a given case that he does not know how to interpret the shadows seen because they are unusual and represent a type of change with which he is not familiar. For instance, a radiologist might be quite familiar with changes produced by tuberculosis and pneumonia and the usual neoplastic growths that occur in the lungs, but might not have had the opportunity of seeing a group of unusual lung tumors and might not be located in a community where silicosis is commonly encountered. Consequently, he would feel no hesitancy in seeking aid when a film presented itself with changes which he did not understand and which he suspected of representing some unusual condition.

We might conclude that the radiologist's place in the diagnosis of lung pathology is that of an important consultant whose findings must be correlated with those of the other medical men. His exact position will vary considerably in different communities and even in the same community, as he deals with different types of referring physicians. He should state his findings and opinions boldly, but he should have no false pride and should frankly state his inability to understand the changes seen in any given case and should seek aid when a group of changes are encountered with which he is not familiar. The radiologists, as well as other medical men, are constantly seeing more early pathology because of the widespread propaganda for routine examinations and because of the more general use of roentgen methods of examination. This fact should stimulate all of us to seek better criteria on which to base an early diagnosis of the unusual lesion which we encounter.

FRANKLIN B. BOGART, M.D.
Chattanooga, Tenn.

COMMUNICATION

TESTIMONIAL DINNER TO DR. I. SETH HIRSCH

On April 17, 1940, in the Hotel Biltmore, New York City, was held a testimonial dinner by the friends and associates of Dr. I. Seth Hirsch. The speakers were Currier McEwen, M.D., Dean of the College of Medicine, New York University; Miles Sturtevant, M.D., of New York City; Harry E. Isaacs, M.D., of New York City; Charles Gottlieb, M.D., of New York City; Henry M. Silver, M.D., of New York City; Arial W. George, M.D., of Boston; Bernard H. Nichols, M.D., of Cleveland, Ohio, President of the Radiological Society of North America; Arthur M. Wright, M.D., of New York City, and A. A. Brill, M.D., of New York City. The Dinner Committee consisted of Charles Gottlieb, M.D., Harry E. Isaacs, M.D., Maxwell H. Poppel, M.D., and Henry K. Taylor, M.D. The Radiological Society of North America was represented by Bernard H. Nichols, M.D., President, and Donald S. Childs, M.D., of Syracuse, N.Y., Secretary-Treasurer. Dr. Hirsch is likewise a member of this Society, as were many of the other guests.

Considerable additional interest was contributed to the occasion by the production of a scene from a play, "The Unlistening Street," written by Dr. Hirsch and Evans Nash. Professor Otto Glasser, Ph.D., of Cleveland, Ohio, spoke wittily on "Roentgen's Discovery in Contemporary Humor." Music was furnished by the Moss String Quartet.

IN MEMORIAM: THOMAS ALLEN GROOVER, M.D.

Thomas Allen Groover was born at Pidcock, in southern Georgia, on May 1, 1877, a son of Thomas Alfred and Sarah Jane (Joiner) Groover. His father, who was a farmer, was a Confederate soldier during the Civil War and had lost an arm in action. Dr. Groover received his early education in the public schools of Brooks County, Georgia. Because of financial considerations it did not seem possible for him to continue his education, but through the good offices of the Hon. Hoke Smith, of Georgia, who was Secretary of the Interior in President Cleveland's Cabinet, he was appointed as assistant messenger in the Department of the Interior. He then came to Washington, in 1893, and resided there all the remainder of his life. In 1894, he entered the Medical Department of Columbian University, which later became The George Washington University, and graduated therefrom in 1898. In 1926, his alma mater conferred upon him the degree of Doctor of Science. After internship at the Garfield Memorial Hospital he was appointed physician with the Isthmian Canal Commission and in that capacity spent the next year in Nicaragua.

Dr. Groover returned to Washington, in 1900, and entered the general practice of medicine. At the same time he began x-ray work at the Central Dispensary and Emergency Hospital. Although he did not completely specialize in radiology until 1912, he was actively engaged in that field from his entry into it, in 1900, up to within three months of his death which occurred on April 20, 1940. It was in those early days from 1900 to about 1904 or 1905 that he suffered the injuries to his hands which resulted in amputation through the left forearm, in 1926, and to final involvement of the right axilla and lung which cost him his life.

Dr. Groover was closely associated with the scientific progress and the organizational development of radiology in this country during the entire first forty years of the present century. He made numerous contributions to the literature and was always interested and quietly helpful in everything that affected the welfare of the specialty that he loved. He was insistent, however, that the radiologist is primarily a physician, and as such he always established a personal relationship with every patient who came under his care. His membership in medical organizations included the following: Medical Society of the District of Columbia, of which he was President in 1925; Fellow of the American Medical Association; Fellow of the American College of Physicians; member of the Southern Medical Association, of which he was Vice-president in 1924; member of the American Roentgen Ray Society and its President in 1925; member of the Radiological Society of North America; Fellow of the American College of Radiology, in which he served both as President and as Chancellor.

Dr. Groover's work as a practising radiologist was characterized by the utmost painstaking care and attention to detail. He not only required the best possible technical work but spared no time nor personal effort to arrive at a correct interpretation and final diagnosis. He had developed in himself to a very high degree those qualities which are indispensable in the good physician—accuracy in observation and a keen sense of relative values. He was a diagnostician of unusual ability. In manner Dr. Groover was quiet and reserved. Being of a very studious nature, he made it a rule to spend at least one hour a day in the reading of medical literature. He had great qualities both of mind and heart which were unusual and outstanding. His ability as an organizer and executive was well known. His careful, methodical attention to all the details of bookkeeping, records, and management laid the foundation for his professional work. He always looked upon business arrangements, however, as a means to more important ends. He was first and last a physician. Even radiology, in every branch of which he was proficient, he insisted must always be looked upon as an integral part of general medical practice. His outstanding mental characteristics were patience and a great capacity for methodical, painstaking care in every diagnostic and therapeutic procedure. His dominant moral characteristic was

The late THOMAS A. GROOVER, M.D.

a keen sense of justice, right, and fair dealing. He had an unfailing patience in compromising differences of opinion among his associates, a sane judgment in arriving at important decisions, and above all, a kindly, tolerant charity toward all with whom he could not agree. He will be greatly missed by his close associates who were accustomed to call upon him almost daily for his helpful counsel, and his loss will be keenly felt in many organizations in which his advice was highly valued. His death adds another martyr to the long roll of those who have sacrificed their lives in the interests of science and humanity.

ARTHUR C. CHRISTIE, M.D.

Washington, D. C.

ABSTRACTS OF CURRENT LITERATURE

CONTENTS BY SUBJECT

THE FOLLOWING ABSTRACTORS HAVE CONTRIBUTED TO THIS ISSUE

BENJAMIN COPLEMAN, M.D., of Perth Amboy, N. J.
JOSEPH T. DANZER, M.D., Oil City, Penna.
SYDNEY J. HAWLEY, M.D., of Danville, Penna.
JOHN B. MCANENY, M.D., of Madison, Wisc.
ANTONIO MAYORAL, M.D., of New Orleans, La.
JOHN M. MILES, M.D., of Lafayette, La.
R. R. NEWELL, M.D., of San Francisco, Calif.
LESTER W. PAUL, M.D., of Madison, Wisc
ERNST A. POHLE, M.D., Ph.D., of Madison, Wisc.
SIMON POLLACK, M.D., of Tulsa, Okla.
ERNST A. SCHMIDT, M.D., of Denver, Colo.
CHARLES G. SUTHERLAND, M.D., of Rochester, Minn.
WILLIS A. WARD, M D., of Chicago

ABSTRACTS IN THIS ISSUE LISTED ALPHABETICALLY BY AUTHORS

LARYNX

Laryngocele. John Blewett. British Jour. Radiol., **12,** 163–167, March, 1939.

At the anterior end of the sinus of the larynx is the small saccule. The thyro-hyoid membrane over the saccule is strengthened at the middle and at the lateral edges but between these places it is weak. The superior laryngeal vessels enter here, which further weakens this spot. When the saccule becomes distended with air, there is little to prevent the formation of a laryngocele. The condition is usually found in those who use the voice excessively or in glass blowers and wind-instrument players. The cyst can remain in the larynx or it may herniate into the neck. Sometimes it appears only on forced phonation and then disappears, but in some cases it may remain and obstruct respiration. Roentgenographic examination has been seldom used in the diagnosis. The appearance of the air-filled tumor on the film is striking, so the diagnosis can be readily made by x-ray examination. An illustrative case is presented.

SYDNEY J. HAWLEY, M.D.

X-ray Examination of Larynx and Pharynx. R. P. O'Bannon. Texas St. Jour. Med., **34,** 849–851, April, 1939.

The radiographic anatomy and physiology of the soft tissues of the neck are reviewed and the value of radiographic study in pathologic conditions pointed out.

A lateral view of the neck is made without preliminary preparation by the use of a soft-tissue technic. Inflammatory or neoplastic diseases of the pharynx or larynx show an alteration in the shape and size of the soft-tissue structures which stand out in contrast to the air in this region.

The information obtained is often greater and more specific than that obtained on clinical inspection. It is also an excellent record of the progress or healing of the diseased condition.

JOHN M. MILES, M.D.

Roentgen Consideration of Lesions in and about the Larynx: Diagnostic Aspects. Adolph Hartung. Illinois Med. Jour., **76,** 125–127, August, 1939.

According to the author, far too little use is made of the x-ray and fluoroscope when considering lesions in and about the larynx—a region which is especially favorable for roentgen examination because of the air passages. Fluoroscopic examination with sagittal and lateral views, with films offering a maximum of soft-tissue contrast, is advised. In some cases opaque media are advantageous. Intralaryngeal films as used by Waldapfel and special procedures such as drawing the larynx forward by means of a curved sound, or over-distending the air passages by the Valsalva method as suggested by Jönnson, are also to be considered. The profession must become more appreciative of the value of these methods, for in some cases they may be the only practicable means of obtaining the necessary information as to the nature, exact location, and extent of a lesion. They should especially be considered in the presence of such complaints as hoarseness, aphonia, difficulty of speech, stridor, recurrent cyanosis, strangling attacks, lump in the throat, dysphagia or pain on swallowing, pain or swelling in anterior part of neck, and suspicion of foreign bodies. The non-radiopaque foreign bodies may cause recognizable soft-tissue changes to reveal their location and a follow-up examination is advisable to rule out the possibility of stricture. Trauma is also an indication, as displacements of parts of the larynx or fractures of the cartilaginous portions may be found, especially if ossification is present.

Inflammatory conditions of the neck, especially retropharyngeal abscess, are to be noted. Conditions which produce narrowing of the lumen of the air passages by external compression, such as post-inflammatory stenosis, thyroid enlargements, tumors, and other swellings in the neck region, may be located.

Tuberculosis, syphilis, benign lesions, tumors of the base of the tongue, lesions of the cervical portion of the esophagus, and neoplasms may be discovered by roentgenographic examination. This is not only an aid in diagnosis, but may suggest the preferable means of treatment, or the site for radon implantation, or topical application of radium. Furthermore, tracheotomy procedures and the proper fitting of tracheotomy tubes may be aided by roentgenographs of the neck. The author pleads for a greater appreciation of the value of these methods, a greater dissemination of the knowledge of such methods, and a more frequent use of them. He reminds us that a chest examination is often incomplete without a detailed examination of the neck, since that is where the trouble may be. Attention is called to the necessity of correlating the clinical findings with the roentgen findings and with those of the laryngologist.

WILLIS A. WARD, M.D.

Roentgen Consideration of Lesions in and about the Larynx. T. J. Wachowski. Illinois Med. Jour., **76,** 128–130, August, 1939.

The author divides the lesions of the larynx which warrant therapeutic irradiation into those of inflammatory and neoplastic origin. For the inflammatory ones, he advises judicious small dosage irradiation, especially when there is perilaryngeal involvement of the phlegmonous type.

For lesions of neoplastic origin, the author states that the benign ones are ordinarily handled by surgery, but in the field of malignant neoplasms there is a great need of advancement of therapy. Irradiation may be applied internally or externally, but the former method has met with little success. The latter method is the one with which we are more concerned and the application may be by either high voltage roentgen rays or the radium bomb. No significant difference in results has been reported thus far.

Irradiation is accepted as the method of choice in all inoperable cases. The accepted rationale of the protracted intensive method is the differential action of

gamma rays on the more undifferentiated cells of the body. It is conceded that the curability of carcinoma of the larynx by irradiation is greatest in the intrinsic lesion and decreases as the growth is removed farther from the vocal cords. Mention is made of a group of radioresistant lesions.

For the technic one must consider the size of the lesion, the age and condition of the patient, and the presence or absence of metastases and infection. If infection is present, it is advisable to precede the main course of therapy by a short period (13 to 26 days) of treatment given in small dosages (5 to 50 r) through large portals. This is intended to control or eliminate the infection and to modify favorably the vasculoconnective tissue. The cellulocidal therapy is best delivered through the smallest portals possible so as to be more likely to irradiate the lesion sufficiently before endangering the general condition of the patient. Round portals are considered better than square ones.

The indications are not clear-cut for either surgery or irradiation, but mention is made of Coutard's classification of laryngeal carcinomas into lesions composed of undifferentiated cells and those composed of differentiated cells. Surgery is recommended for the differentiated type since cure by irradiation is considered almost impossible. But in the undifferentiated group, irradiation therapy is the method of choice in all cases, since it is never biologically operable because of early and widespread metastasis. If surgery should be insisted upon, pre-operative irradiation is advised.

The main course of irradiation should be planned so as to deliver the calculated dose in about 25 or 30 days. Protraction of therapy over more than 60 to 90 days is to be guarded against because this introduces the risk of "radio-vaccination," a condition of increased radioresistance of the tumor cells. Most of the successfully treated cases, in the author's experience, using portals of average size (50 sq. cm. ±), fell within a certain dosage zone, usually 4,000 r × 2 (measured in air).

WILLIS A. WARD, M.D.

The Treatment of Cancer of the Larynx, with Comparison of Results Obtained by Surgery and Radiation Therapy. M. F. Arbuckle. Southern Med. Jour., **32,** 1008–1012, October, 1939.

Deaths from carcinoma of the larynx constitute 1.8 per cent of all cancer deaths in the United States. The treatment to be employed varies with the location, extent, and degree of malignancy. Authorities differ as to the form of therapy most advisable.

The site of origin of laryngeal carcinoma most often is the true vocal cord and, since metastases do not occur while the tumor is confined to the cord, surgical removal of cordal cancer results in a lasting cure in 80 per cent of cases, with little inconvenience, low mortality, and no loss of function. Irradiation is less satisfactory in this type of cancer than is surgery.

In other types of laryngeal cancer with lymphatic involvement the results of radiation therapy alone are better than those obtained by surgical treatment. It is a mistaken idea, however, that suffering, mutilation, and danger to life are greater from surgery than from irradiation, particularly when the radiation is administered to the syphilitic, the aged, or the infirm.

The results of 34 cases of carcinoma of the larynx are tabulated, and a typical case of intrinsic carcinoma and one of an extrinsic carcinoma of the larynx are reported in detail.

JOHN M. MILES, M.D.

LEUKEMIA

Roentgen Therapy of Leukemia with Variable Filters. A. Possati. Strahlentherapie, **65,** 54, 1939.

In 1925, Ghilarducci recommended a technic of roentgen irradiation with variation of filters during a course of treatments. The author has used a modification of this plan in cases of leukemia. He employed from 180 to 200 kv., 2 ma. and doses of one-thirtieth to one-tenth S.E.D. The fractional doses are given with the filter with 1 mm. Al, with 3 mm. Al, with 5 mm. Al, and then with 0.5 mm. Zn + 3 mm. Al, exposing two large fields, the liver and spleen. Often Possati saw striking response even in late stages which had become fairly radioresistant, and he proposes, therefore, to give this method a trial in the treatment of deep-seated malignant neoplasms.

ERNST A. POHLE, M.D., Ph.D.

The Symptomatology of Lymphoma: Its Endless Variety. J. H. Means. Jour. Am. Med. Assn., **113,** 646–649, Aug. 19, 1939.

Lymphoma is a disease both ubiquitous and pleomorphic, presenting itself in ever-changing symptomatology. It has a great capacity for producing bizarre clinical pictures and a propensity for being heralded by signs and symptoms diagnostically misleading.

The disease is generally considered to be neoplastic—a neoplasm taking origin from a cell of the lymphocytic series anywhere between the primordial, or stem cell, and the mature cellular elements. Pathologists recognize several types described by such terms as lymphoblastoma, reticulum-cell sarcoma, lymphosarcoma, Hodgkin's disease and giant follicular lymphoma. Whether these types represent separate entities or merely varieties of a single entity is undecided. That in certain cases the pathologic picture may pass from one type to another suggests that the disease is fundamentally unitary.

All types are, relatively speaking, radiosensitive and yield to some extent for a time to rather low dosage of radiation.

It has been shown that rate of progress of the disease bears some relation to the anatomic picture. Life expectancy is longer when the anatomic picture called "giant follicular lymphoma" is present than in any of the others. That type is seemingly the least malignant.

Although fundamentally neoplastic, the disease lymphoma resembles more closely in its clinical course and behavior certain chronic infections than it does other malignant tumors. While it may start locally in

one particular lymph node or other tissue where cells of the lymphatic series are present, its inherent tendency is to become systemic or even constitutional. In most cases, at some stage of the disease, the course is febrile.

The symptoms of the disease may be the result of local interference with the function of involved organs or of pressure on neighboring ones, or of the general effects of the systemic extension of the lymphomatous process, such as fever, fatigue, or itching. Since almost any part, or parts, of the body may be involved, the symptoms and signs which may be produced are nearly endless.

The absolute diagnosis of lymphoma can be made only by biopsy. A negative biopsy does not exclude the existence of lymphoma in the patient. The x-ray appearance of intrathoracic or gastro-intestinal lesions sometimes gives almost uncontrovertible evidence, and the rapid melting of a lesion under x-radiation leaves only a very small, if any, doubt as to its lymphomatous nature. Subsidence of fever under such treatment has a similar significance.

CHARLES G. SUTHERLAND, M.D.

THE LUNGS

Hydatid Cysts in Both Lungs. H. Kenrick Christie. Australian and New Zealand Jour. Surg., **8,** 373–378, April, 1939.

In reviewing the records of the Australasian Hydatid Registry, of 1,350 cases, the author collected 250 cases of lung cysts, of which 16 were bilateral. He discusses in detail the symptomatology, surgical treatment, and prognosis of this rare condition and presents numerous excellent radiographs to illustrate his case reports. The similarity between hydatid cysts of the lung and large metastatic tumors (such as metastatic hypernephroid carcinoma) is striking.

SIMON POLLACK, M.D.

Transitory Lung Infiltrations Associated with Eosinophilia (Löffler's Syndrome): Report of a Case. D. C. Wharton Smith and Alexander J. Alexander. Southern Med. Jour., **32,** 267–272, March, 1939.

An atypical and controversial case is reported, characterized by diffuse transitory areas of density in the lungs (lasting only 14 days), eosinophilia, acute illness, and death, in two months, of a seven-year-old white girl. A complete autopsy report is included. The diagnosis is unsettled because of the complicating features. Löffler's syndrome, septicemia, and leukemia are considered.

JOHN M. MILES, M.D.

Bronchographic Study of Apparently Healed Lung Abscesses. R. Maxwell Franklin. Am. Jour. Med. Sci., **198,** 95–98, July, 1939.

Bronchographic study after the disappearance of the symptoms and the roentgenographic shadows of acute suppurative bronchopulmonary disease often reveals residual pathologic defects in the lungs and bronchi that are otherwise unrecognizable.

Bronchography should be used routinely to determine the status of the bronchi and lungs after the symptoms and roentgenographic shadows of the acute suppurative process have disappeared.

The clinical concept of healing of acute suppurative bronchopulmonary processes should be broadened to include the residual pathologic changes in the lungs and bronchi.

BENJAMIN COPLEMAN, M.D.

METASTASIS

Skeletal Metastasis in Cancer. Charles F. Geschickter and I. H. Maseritz. Jour. Bone and Joint Surg., **11,** 314–322, April, 1939.

These authors have reviewed 5,739 cases of carcinoma arising from various organs and have recorded the incidence and location of metastasis to bone as found on x-ray examination. Of this number, 356 showed metastasis to bone, and in 60 cases (16.8 per cent) the primary site of the growth was not located. Pathologic fractures occurred in 19 instances (31.6 per cent); solitary lesions occurred in 34 (56.6 per cent).

Metastatic hypernephroma occurred in 41.5 per cent; bone metastasis from breast cancer, 5.6 per cent; in cancer of the prostate, 13 per cent; urinary bladder cancer, 47 per cent; one in 80 cases of carcinoma of the esophagus showed bone metastasis; seven of 750 cases of carcinoma of the stomach; three of 620 cases of carcinoma of the large intestine and rectum; one in 46 cases of carcinoma of the liver; six of 76 cases of carcinoma of the thyroid; five in 86 cases of carcinoma of the uterus.

The spine and pelvis are the most frequent sites of bone metastasis, but other bones may also be involved. Irradiation of metastatic lesions relieves pain and prolongs life.

J. B. MCANENY, M.D.

Metastasis to Bone from Carcinoma of the Gastro-intestinal Tract. Ralph K. Ghormley and Jorge E. Valls. Jour. Bone and Joint Surg., **21,** 74–78, January, 1939.

Review of the pertinent literature reveals that autopsy studies place the figure of bone metastasis from gastro-intestinal carcinoma too high, because of the terminal state of the disease.

Most metastases are seen in the spine, pelvis, and ribs, the long bones being but rarely involved. The incidence of metastasis to bone is found to be between 0.2 and 0.5 per cent of cases.

JOHN B. MCANENY, M.D.

PELVIC MEASUREMENTS

Stereoscopic Measurements of Distances in the Female Pelvis and on the Fetal Skull. Benedict Westergaard. Acta Radiol., **20,** 33–39, February, 1939.

The author reports his experience and findings in stereoscopic measurements of the maternal pelvis and the fetal head. From frontal roentgenographs, the su-

perior transverse diameter and the distance between the ischial spines were determined, occasionally also a minimum value of the conjugata vera. However, the conjugata vera was, as a rule, determined from profile roentgenographs. As far as the fetal head is concerned, its biparietal and bitemporal diameters can be ascertained only when they lie at right-angles to the central rays from the two foci. The stereoscopic measurements of the conjugata vera were less accurate than those of the fetal skull. While in the latter case the error amounted to less than 1 mm., the difference between roentgen measurements of the conjugata vera and check measurements taken by the direct method during laparotomy was higher, though never exceeding 3.4 mm.

ERNST A. SCHMIDT, M.D.

Pelviradiography and Clinical Pelvimetry: Comparative Values in the Prognosis of the Outcome of Labor. Emanuel M. Rappaport and Samuel J. Scadron. Jour. Am. Med. Assn., **112,** 2492–2497, June 17, 1939.

X-ray examinations were originally employed for the purpose of attesting fetal presence, multiple fetus, and fetal deformities. During the last fifteen years several methods of x-ray pelvimetry have been introduced.

This study covers a series of 350 patients, the x-ray examination being made ten days prior to the calculated date of labor. In instances of miscalculation, when labor was delayed several weeks, x-ray examination was repeated at term.

In the classification of the various types of pelves, such factors as size of inlet, relationship between head and inlet, sacrosciatic notch, inclination of sacrum and lateral bone, sacral promontory, subpubic angle, shape of arch, length of rami and side walls of the pelvis were taken into consideration. The classification adopted was that of Caldwell and Moloy. In the series studied, the percentages of the various major pelvic types closely approximated those of other observers, with the exception of the platypelloid type.

The gynecoid pelvis is the type most frequently encountered in any series; it occurred in 158 of these 350 cases. The anthrapoid type was next in frequency, with 52 cases. Forty-four women in this series showed the true android type of pelvis, and 23 the mixed android type (*i.e.*, with android posterior segments and gynecoid forepelvis). There were 12 cases of true platypelloid pelvis and nine cases of mixed type. The authors explained the more frequent occurrence of this type in their series by the fact that their patients were entirely those of the Jewish race, wherein females of the hyposthenic and pyknic type are frequently encountered.

The types were charted to indicate the spontaneous deliveries, the elective forceps deliveries, and the instrumental deliveries in each.

The authors felt that clinical experience without the use of pelviradiography has the advantage over the use of x-ray interpretation alone, and that it was of paramount importance that the obstetrician develop himself clinically rather than to rely solely on x-ray methods. The greatest acumen in determining the relative size of the passage and passenger and in selecting the appropriate operative procedure is best obtained when the experienced obstetrician combines his clinical knowledge with pelviradiography.

CHARLES G. SUTHERLAND, M.D.

PEPTIC ULCER

A Frequent Non-ulcerous Cause of the Peptic Ulcer Syndrome. Lester M. Morrison, William A. Swalm, and Chevalier L. Jackson. Pennsylvania Med. Jour., **43,** 243–247, December, 1939.

Gastritis may be the only pathology found in 30 per cent of patients exhibiting the textbook picture of peptic ulcer. The symptoms of gastritis are so variable that they may range from complete absence of symptoms to the perfect mimicking of peptic ulcer with its food or alkali relief, and hematemesis. Deformity of the duodenal cap has been seen on x-ray examination, which, at operation, has proven to be due entirely to gastritis.

JOSEPH T. DANZER, M.D.

Ulcerative Gastritis and Residual Lesions. H. E. Robertson. Jour. Am. Med. Assn., **112,** 22–27, Jan. 7, 1939.

The stomach represents a very complex physiologic mechanism affected almost as frequently and as severely by extrinsic as by intrinsic factors. When an extrinsic influence, such as a lesion in the brain or a severe anemia, upsets this mechanism the resulting disturbances may in turn bring about local changes. If one injects the arterial trunk supplying the gastric mucosa with any substance which permits the capillaries to be visualized, they will appear in the mucous membrane as an extremely thick network so numerous that, by dilating them with India ink, one will see that the surface resembles a solid mass of this black substance. Whenever, for any reason, an abnormal congestion of these capillaries occurs, one receives the same impression of a tremendously rich capillary blood supply to the entire mucosa. In ordinary microscopic preparations, even of freshly preserved normal stomachs, these capillaries are but inadequately seen, but their importance in the interpretation of many lesions in the stomach cannot be overestimated.

Apparently whenever congestion of the capillary network occurs, even with the normal physiologic stimuli of digestion, a slight over-distention in any local area may bring about a rupture of one or more capillary loops. As soon as the hemorrhage occurs, necrosis of the involved part is an inevitable result. This necrosis is promptly digested and leaves an ulcer, which ordinarily is promptly repaired with complete restoration of the integrity of the mucosa. In most instances the entire process is without a single discoverable clinical sign or symptom.

While many small hemorrhages and their resulting ulcers may be followed by complete restitution, often there remain more or less definite gross or microscopic evidences of the fact that, after one or more local areas

in the stomach have previously suffered from ulcers, imperfect instead of perfect healing has resulted.

When hemorrhage in the mucosa produces necrosis, the production of mucin in that area ceases and the necrotic area is promptly digested until the living glands are exposed and fresh mucous reserves can once more inhibit the action of the digestive ferments. The lack of sufficient mucin as to either quality or quantity may thus account for the failure of proper or prompt healing of certain ulcers. The possibility that some nervous mechanism, definitely affected by cerebral conditions, may play a rôle in the elaboration of this substance has to be seriously considered.

When ulcers are deeper and more severe, after healing, a stellate scar may persist as a residual gross lesion. Atrophic processes or hyperplastic changes may result. Under certain circumstances hyperplastic changes in the mucous glandular cells may proceed to the stage of autonomous newgrowth. Carcinomas probably most commonly have their origin in atrophies, disorganizations and reparative hyperplasias which represent the residuals of many previous ulcers.

The clinician should not use the almost meaningless term "chronic gastritis." Instead, he should employ names which have a definite significance such as atrophy of gastric mucosa, hyperplasia of gastric mucosa (localized or polypoid), ulcerative gastritis, syphilitic, tuberculous, or alcoholic gastritis, atony of the stomach or achlorhydria.

CHARLES G. SUTHERLAND, M.D.

Chronic Peptic Ulcers in Children. Howard B. Kellogg. Northwest Med., **38**, 129–131, April, 1939.

Kellogg was able to find only 36 reported cases of chronic peptic ulcer in children, adding to this number one of his own. The pathology of these ulcers is similar to that of ulcers found in adults. The location, however, seems to be the reverse. In all these cases, 21 were located in the stomach and 15 in the duodenum.

The symptoms are somewhat less typical in children and may be accompanied by rather alarming conditions, such as severe hemorrhage or perforation. The author states that a large proportion of the patients present a varying degree of pyloric obstruction.

If the possibility of the lesion is suspected, the writer believes that these ulcers will be found more frequently among children.

A. MAYORAL, M.D.

Anterior Pituitary Tumor Associated with Cachexia, Hypoglycemia, and Duodenal Ulcer. Maurice P. Foley, Albert M. Snell, and Winchell M. Craig. Am. Jour. Med. Sci., **198**, 1–8, July, 1939.

A case is reported in which a curious aggregation of symptoms, including persistent hypoglycemia, symptoms of cachexia hypophyseopriva, lowered metabolism, an eunuchoid status, and an active and incapacitating duodenal ulcer, was encountered in association with a tumor of the anterior lobe of the pituitary body, which proved, microscopically, to be a chromophobe adenoma.

Several other investigators have reported peptic ulceration associated with intracranial lesions since Rokitansky first suggested the neurogenic origin of gastro-intestinal ulceration in the early 1840's. Briefly, there is clinical and experimental evidence to suggest that acute and chronic peptic ulcers are among the changes produced in the gastro-intestinal tract by intracranial lesions.

The severe hypoglycemia, with the absence of referable symptoms, the lack of response to administration of glucose and epinephrine, and the persistence of the low blood sugar even after removal of a portion of the tumor are noteworthy.

The lowered metabolic rate, common in pituitary cachexia, may represent not only an endocrine effect but secondary changes in nutrition.

BENJAMIN COPLEMAN, M.D.

PNEUMONIA

Lipoid-cell Pneumonia: Adult Type. Henry M. Thomas, Jr., and William F. Rienhoff, Jr. Southern Med. Jour., **32**, 1077–1080, November, 1939.

A case of lipoid-cell pneumonia in a 35-year-old white man is reported. This condition is important because it may simulate a tumor of the lung. When suspected, the diagnosis can be confirmed by obtaining the history of the excessive use of oily nasal sprays or drops and by finding lipoid cells in the sputum. In this case, a medicated oily nasal spray had been used for over three years. The difficulty of diagnosis is shown by the fact that this case was mistaken clinically, at operation and at the autopsy table, for a tumor of the lung. Microscopically, there was an interstitial fibrosis with fat droplets in the alveoli, alveolar walls, and lymph nodes.

JOHN M. MILES, M.D.

Lipoid Pneumonia: Report of a Case. Anna M. Young, H. S. Applebaum, and P. B. Wasserman. Jour. Am. Med. Assn., **112**, 2406–2409, June 10, 1939.

Lipoid pneumonia (referred to by various authors as oil pneumonia, oil aspiration pneumonia, oil inspiration pneumonia, paraffinoma, paraffin oil tumor, and pneumonolipoidosis) occurs most frequently in infants and in the debilitated old, resulting commonly from the administration of laxative oils or the therapeutic instillation of oils into the nasopharynx with subsequent aspiration of the oily or fatty substance into the lung. Less frequently this lesion has been reported in adults not necessarily debilitated. The authors report a case of the adult type (women, aged 42 years) following repeated aspirations of animal oil, probably cod liver oil, taken by mouth. In addition to the conditions found in the lung similar to those reported by others, metastatic deposits of lipoid material were found, principally in the kidneys and spleen, together with an associated necrotizing arteritis and peri-arteritis.

The lipoid material present in the tissues in this case stained with osmic acid and was saponifiable. Liquid petrolatum stains with scarlet red but not with osmic

acid and is not saponifiable. Animal oil and animal fat (milk, cream, cod liver oil, rabbit fat, and lard oil) produce more severe lesions than do mineral oils. The roentgenogram in this case exhibited the characteristic image of an ordinary pneumonia and not the increased linear markings (fibrosis) or perihilar shadow commonly observed from the aspiration of mineral oil.

CHARLES G. SUTHERLAND, M.D.

RADIATION

Electron Diffraction Studies of Thin Films. I.—Structure of Very Thin Films. L. H. Germer. Phys. Rev., **56,** 58–71, July 1, 1939.

Three pages of diffraction rings from evaporated films of some metals and salts are shown in this article. Their interest to the radiologist is the similarity to diffraction rings by x-rays. The wave aspect of the electron has become as commonplace an idea as the particle aspect of light and x-rays. These diffraction patterns by the author, who is a pioneer in electron diffraction, are worth a radiologist's attention, not only for their philosophical import, but for their beauty.

In the experiment related, the author was using them to discover the crystalline structure of very thin evaporated films (2 Å. average thickness to several hundred Å.).

R. R. NEWELL, M.D.

The "Quality" of High Voltage Radiations. Part II.—"Quality" within a Scattering Medium. J. R. Clarkson and W. V. Mayneord. British Jour. Radiol., **12,** 168–180, March, 1939.

The authors have measured the effective wave length and the changes in effective wave length which occur in a scattering medium under various conditions. Two methods were used, an emission and an absorption method. The emission method, which is the more accurate, depends upon the difference in photo-electric emission from the walls of metal and carbon ionization chambers with changing wave length; the absorption method depends on the difference in absorption of different wave lengths. The apparatus and procedures are described in detail.

The effective wave length is longer for larger fields and is longer at depths of from 10 to 15 cm. It may be increased to as much as twice the length of the primary. The radiation at the edge of a large field shows half the softening observed in the center. For very small fields this difference is much diminished. These changes are the result of differences in the ratio of scattered to the total radiation. Measurements were made to determine how much of the total radiation was scattered radiation and how much primary at various depths. The absolute amount of scattered radiation reaches the maximum between 2 and 5 cm. below the surface, depending on the wave length of the primary. In general, the scattered radiation makes up about 80 per cent of the total radiation at 14 cm. depth. Variations in the effective wave length of the primary, filtered through water at such a distance that the scattering was negligible, were measured.

The results are presented in the form of graphs. These findings have an important bearing on the interpretation of the selective action of different wave lengths.

SYDNEY J. HAWLEY, M.D.

RADIUM

The Use of Radium in Maintaining a Patent Frontonasal Opening in External Operations of the Fronto-ethmoid Group of Sinuses: A Preliminary Report. Henry L. Williams and Robert E. Fricke. Ann. Otol., Rhinol., and Laryngol., **48,** 412–418, June, 1939.

Difficulty in maintaining a patent frontonasal opening is a frequent cause of failure in external operations on the fronto-ethmoid group of sinuses. The authors tried the application of radium post-operatively in 20 cases with apparently good results. The radium was used in the form of a 50-mg. applicator filtered by 1 mm. of brass, 0.5 mm. of silver, and 2 mm. of rubber, for a total dose of 9 gram-minutes. Although the results in this small series of cases were satisfactory, a review of cases not treated by radium seems to indicate that adequate technic in performing the operation is more important than any other factor.

LESTER W. PAUL, M.D.

Individualized Radium Therapy of Carcinoma of the Cervix. H. Reichenmiller. Strahlentherapie, **65,** 595, 1939.

The author contends that the success of radiation therapy of carcinoma of the cervix depends largely on the effect of radium. The recurrences can be explained by the rapid drop of radiant energy from the radium applicator toward the periphery. He groups the failures under three headings: cervix cavity carcinoma, in which the tumor grows within the canal leaving the os intact, the huge cervical cancer, and the "spina" recurrence, a carcinomatous mass which develops near the spina ischiadica after the local growth has been successfully destroyed. Reichenmiller outlines his modifications of the standard treatment method which permits higher doses in the periphery, thus affecting cancer cells which otherwise would not receive sufficient dosage. By cauterizing some of the central tumor mass first, the radium screens can be placed closer to the periphery, and, by using longer radium applicators which extend 1 cm. beyond the cervical os, the end-results can be improved definitely.

ERNST A. POHLE, M.D., Ph.D

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AUTHORS